TM 8-285

U. S. WAR DEPARTMENT

TECHNICAL MANUAL

TREATMENT OF CASUALTIES FROM CHEMICAL AGENTS

July 10, 1941

TECHNICAL MANUAL

TREATMENT OF CASUALTIES FROM CHEMICAL AGENTS

CHANGES No. 1

WAR DEPARTMENT,
WASHINGTON, February 14, 1942.

TM 8–285, July 10, 1941, is changed as follows:

9. Mustard (HS).

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d. Prophylaxis.—To be effective, prophylactic measures must be instituted * * * applied to absorb any mustard remaining on the skin. Next, the area is dabbed gently and repeatedly with sponges dampened with **a solution of dichloramine-T in triacetin (see par. 5*j*, app.), or if this solution is not available with** gasoline, kerosene, carbon tetrachloride, or alcohol. The skin surface within and beyond the outlines of the contaminated area is then vigorously scrubbed with soap and water and then the area is patted dry. The sponging materials contaminated in this process must be burned or buried. The protective ointment also removes mustard effectively from the skin surface if it is applied with rubbing and then wiped off. **The application of amyl salicylate daily for several days helps lessen edema and gives an immediate analgesic effect. The application of amyl salicylate is made either by spraying or by dipping gauze into the drug and applying this gauze to the lesion. The use of triple dye (see par. 5*l*, app.) after the first few days helps maintain asepsis and promotes rapid healing.** Products containing active chlorine such as * * * against bronchitis and the development of broncho-pneumonia.

e. Immediate treatment.—(1) With the development of erythema the skin should first be treated with solvents by the careful technic described above. If chlorinating agents are used they will aggravate the erythema. **Protective ointment should not be used *after* the erythema appears as it will increase the irritation.** Very little else can be done * * * has been found to be effective.

(2) If the eyes are involved they must be irrigated continuously for some hours with **one of the following solutions:** 2 percent sodium bicarbonate, **normal saline, 2 percent boric acid solution, or chloramine-T 0.5 percent in either distilled water or normal saline solution. If necessary a solution of neosynephrin HCL, pontocain HCL, and boric acid (see par. 5*k*, app.) may be instilled to relieve pain. *No cocaine* should be used due to the fact that it causes corneal irritation. The eyes should not be bandaged due to the danger of pressure necrosis of the cornea.**

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Dark glasses or a brown paper eyeshade (one which shades the eye from above and not a so-called shade which completely covers the eye) may be worn to give relief from photophobia.

* * * * * * *

f. Further treatment.

* * * * * * *

(2) If the eyes are seriously involved, solutions noted above may be used to control pain. Cocaine must *not* be used. In severe conjunctival or corneal cases instill one of the following drops or ointments three times daily: liquid paraffin, cod liver oil made alkaline with sodium bicarbonate, glycerin, or acriflavine in castor oil 1:15,000. In those cases with corneal involvement or marked photophobia, instillation of atropine 1 percent solution or atropine ointment is made every day until the symptoms of iritis subside. Dark glasses or brown eyeshades should be worn until the keratitis and iritis subsides. Dark glasses should be removed as soon as possible to prevent neurasthenic symptoms. If the eye discharge becomes purulent, colloidal silver irrigations (argyrol 10 percent) followed by boric acid solution should be used. In severe cases of secondary infection the use of sulfonamide therapy or symptomatic use of nonspecific protein or heat therapy should be tried.

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[A. G. 062.11 (2–3–42).] (C 1, Feb. 14, 1942.)

10. Lewisite (M–1).

* * * * * * *

d. Prophylaxis.—Prophylaxis must be instituted within 1 minute after exposure to be really effective against liquid lewisite. Contaminated clothing must be quickly removed with the usual precautions, and, if possible, treatment should be started at the same time. The exposed areas should be repeatedly swabbed with hydrogen peroxide. This may be used in 8 percent solution (30 percent superol of Merck diluted to 8 percent hydrogen peroxide). If this solution is not available hydrogen peroxide USP 3 percent may be substituted. Fresh swabs should be used for each application and should be destroyed as soon as used. If peroxide is not available the contaminated skin areas should be swabbed alternately and repeatedly with sodium hydroxide solution and with alcohol. * * * the skin should be thoroughly washed with soap and water.

e. Immediate treatment.—The first procedure is to apply the prophylactic measures described above. **For burns from lewisite vapor** sometimes all that can be done is to attempt to relieve the itching, by the use of simple dressings or by the application of antipruritic ointments, as recommended for mustard (par. 9*e*). **While the protective ointment M–1 affords protection against lewisite if applied before contamination, this ointment should not be used on the skin for the treatment of lewisite.** When burns are produced by liquid lewisite the most effective treatment up to 24 hours * * * These include adequate fluid intake and output, intravenous glucose, high carbohydrate diet, and vitamin mixtures. **The symptoms of lewisite burns of the *eye* appear more quickly and are generally more painful and destructive than those produced by mustard. In addition the wound excretions contain arsenic. Any treatment, therefore, must be prompt, and irrigations must be especially thorough and long continued. The treatment is the same as for mustard except that chloramine-T or solutions containing free chlorine are not to be used. Solutions of potassium permanganate up to 0.5 percent and hydrogen peroxide up to 0.5 percent may be used with caution for the initial irrigation followed by water or normal saline.**

*f. **Later** treatment.*—**Blisters may be treated by draining and removal of the cover. It must be kept in mind that the blister fluid is vesicant and that precautions must be taken to protect surrounding areas. The later treatment does not differ from deep burns due to other causes.** General as well as local treatment * * * adjunct to the usual treatment of this condition if it develops.

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[A. G. 062.11 (2–3–42).] (C 1, Feb. 14, 1942.)

APPENDIX

1. Tabulated data on certain chemical agents.

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VESICANTS

Name CWS symbol	MUSTARD (Bisbetachlorethysulfide; "Yperite" "Senfgas") HS	LEWISITE (Betachlorvinyldichlorarsine) M-1	• • •
*	* * *	* *	*
First-aid treatment.	*Skin*: If liquid, remove excess with cloth sponge. Wash with **a solution of dichloramine-T in triacetin. If this is not available wash with** alcohol, kerosene, or carbon tetrachloride, or hot water and soap. Apply bleach paste or protective ointment and remove by wiping or washing. Treat burns as ordinary heat burns. *Eyes*: Irrigate with 2 percent sodium bicarbonate or saturated boric acid. Instill 2 percent butyn sulfate **or neosynephrin HCL, pontocain HCL, and boric acid solution** for pain. Do not cover or bandage.	*Skin*: If liquid, remove excess with cloth sponge. Wash **repeatedly with hydrogen peroxide. If this is not available wash** with soap and water, then with sodium hydroxide solutions *followed by water*. If large skin areas are involved, the skin should be excised. Multiple incision with cupping may be a valuable procedure. *Eyes*: Same treatment as for mustard injuries.	* * *
Protective measures.	Service gas mask; protective clothing; protective ointment; collective protection.		

LACRIMATORS

Name CWS symbol	TEAR GAS SOLUTION (Chloracetophenone, chlorpicrin, chloroform) CNS	CHLORACETOPHENONE CN	BROMBENZYLCYANIDE CA	* * *
	* *	* *	* * *	
First-aid treatment.	Face wind with eyes open; in more severe cases, wash eyes with **an aqueous solution of sodium sulfite or with a 2 percent solution of sodium bicarbonate.** ***Do not rub or bandage eyes.*** Persistent pain may be relieved by instilling 2 percent butyn sulfate, **or neosynephrin HCL, pontocain HCL, and boric acid solution.** If necessary, skin may be washed with **an alcoholic sodium sulfite solution or with a solution of sodium bicarbonate.**			* * *
Protective measures.	Service gas mask; collective protection.			

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[A. G. 062.11 (2-3-42).] (C 1, Feb. 14, 1942.)

5. Formulary.—*a. Sodium bicarbonate solution, 2 percent.*—(1) This can be approximated by dissolving 1 teaspoonful of Sodium bicarbonate in 1 glass of water.

(2) This solution is used as a wash for the eyes, buccal, and nasopharyngeal cavities, following contamination by vesicant gases.

b. Alkali solution for prophylaxis against lewisite.—

	Gm. or c. c.
Sodium hyydroxide	10.0
Glycerine	30.0
Water, distilled, q. s. ad	100.0

The contaminated skin areas should be swabbed alternately and repeatedly with sodium hydroxide solution and with alcohol. *This solution to be used only if hydrogen peroxide is not available.*

c. Compound tannic acid powder.—* * *

* * * * * * *

(3) The tannic acid solution may be applied by sponging or spraying, followed after 15 minutes by 10 percent silver nitrate solution used in the same manner, this sequence being repeated as often as necessary until a firm eschar is formed.

* * * * * * *

e. Antipruritic ointment No. 88.

	Gm. or c. c.
Benzyl alcohol	50. 0
Stearic acid	30. 0
Glycerine	10. 0
Alcohol	8. 0
Pontocain	1. 0
Menthol	1. 0

Dissolve the **pontocain** and menthol in the alcohol and the benzyl alcohol, and **add the glycerine.** Place the stearic acid in a casserole, and liquefy at a temperature just below the melting point by the gentle application of heat. Add the **warmed** liquid mixture slowly to the melted stearic acid, stirring continuously with a rod or pestle until gradual cooling results in solidification of the mixture. Keep in air-tight ointment jars or collapsible tubes.

f. Protective ointment, CWS, issue.—For protective and prophylactic action on the skin against vesicants.

(1) As a preventive, apply liberally to unprotected parts of the body before exposure to vesicant gases. Leave on skin.

(2) For first aid, apply as soon after exposure as possible. Rub in vigorously for at least 1 minute. Remove after 5 minutes; then apply a second coating, rub in, and leave on the skin.

* * * * * * *

h. Cod liver oil ointment.

	Gm. or c. c.
Cod liver oil	50. 0
Petrolatum	50. 0

(1) Sterilize the petrolatum in an autoclave, cool, and mix thoroughly with cod liver oil. **Sodium bicarbonate should be added to render the ointment alkaline (approximately 1 gm. needed).** If desired, small amounts of turpentine or tincture of myrrh may be incorporated to mask the odor of cod liver oil.

(2) To be used especially with burns where the healing is sluggish.

i. Aqueous sodium sulfite solution.

	Gm. or c. c.
Sodium sulfite	0. 4
Water, distilled	25. 0
Glycerine	75. 0

(1) Dissolve the sodium sulfite in the water and add the glycerine.

(2) To be used as eye drops for relief of symptoms from lacrimators.

j. Dichloramine–T in triacetin.

	Gm. or c. c.
Dichloramine–T	20. 0
Triacetin	80. 0

(1) Dichloramine–T and triacetin used in this solution to meet specifications of the Medical Department, U. S. Army.

(2) After the mustard has been grossly removed from the skin the areas are dabbed gently and repeatedly with sponges dampened with the dichloramine–T in triacetin solution.

k. Lotion for eyes and nose.

	Gm. or c. c.
Neosynephrin HCL, 1 percent	25. 00
Pontocain	0. 25
Boric acid, saturated solution	75. 00

To be used as drops in the eye to relieve pain.

l. Triple dye.

Brilliant green	1:400
Gentian violet	1:400
Acriflavine, neutral	1:1000

(1) A mixture is made of equal parts of the dyes.

(2) This solution is applied to the burned area to form a protective eschar.

[A. G. 062.11 (2–3–42).] (C 1, Feb. 14, 1942.)

BY ORDER OF THE SECRETARY OF WAR:

G. C. MARSHALL,
Chief of Staff.

OFFICIAL:

E. S. ADAMS,
Major General,
The Adjutant General.

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TECHNICAL MANUAL

TREATMENT OF CASUALTIES FROM CHEMICAL AGENTS

CHANGES No. 2

WAR DEPARTMENT,
WASHINGTON, June 9, 1942.

TM 8–285, July 10, 1941, is changed as follows:

SECTION XIII (ADDED)

PROPHYLAXIS AND TREATMENT OF ANIMAL CASUALTIES FROM CHEMICAL WARFARE AGENTS

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42. General.—*a.* The following information is intended to provide instruction to Medical Department personnel relative to the pathology, symptoms, prophylaxis, and treatment of animal casualties resulting from exposure to chemical agents.

b. Animals, particularly horses, do not differ materially from men in susceptibility to chemical agents. However, the horse and mule show a relative immunity to the effects of the irritant smokes and to the lacrimators under ordinary field conditions. Variations between man and animal casualties will be particularly stressed.

43. Lung irritants.—*a. General.*—The primary pulmonary irritants are phosgene, chlorpicrin, and chlorine. With all lung irritants a latent period of from 1 to 12 hours or more may intervene between exposure and the onset of any detectable symptoms, but it may be absent or very short after exposure to high concentrations of chlorine. Because of the latent period it may be difficult at times to determine if exposure has actually occurred. Questionable animal cases should be given the benefit of any doubt and kept at rest for 24 hours. Pulmonary edema is the most serious effect of pulmonary irritants. The condition is produced essentially by irritation of the walls of the bronchioles, alveoli, and the adjacent capillary endothelium, and results in a tremendous outpouring of fluid into the alveoli.

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Clinically this is manifested by decreased expansibility of the lungs, increases in density which may not be revealed by changes in the percussion note or by transmission of breath sounds, and also by the presence of moisture indicated by râles of varying types. Physiologically, this pulmonary edema interferes very seriously with the gaseous exchange in the lung and so contributes to the development of an anoxemia. Moreover, the edema plus the local changes in the blood vessel wall, including thrombosis, and the increased viscosity of the blood, all interfere with the circulation through the lung and add to the anoxemia. If this interference with the circulation is maintained, general cardiac collapse may ensue.

b. Phosgene (CG).—(1) *General.*—Phosgene is a colorless gas at ordinary temperatures and pressures, and is heavier than air. It is nonpersistent and protection against it is not ordinarily necessary after 10 minutes. It has an odor of new cut hay or ensilage.

(2) *Pathology.*—Relatively high concentrations of phosgene may be breathed without immediate irritation of the respiratory tract, hence the most distant bronchioles and alveoli of the lung are exposed to the gas. In contact with moisture of the alveolar walls, the gas hydrolizes, producing hydrochloric acid. The irritant effect is supposed, but not proved, to be due to the action of the hydrochloric acid thus formed. As a result of this irritation the permeability of the alveolar and capillary walls is increased, causing a pulmonary edema. There are relatively few changes in the upper respiratory tract.

(3) *Symptoms.*—Actually there may be no respiratory symptoms of any sort until pulmonary edema develops suddenly and dramatically. Sometimes the latent period may last for as long as 12 to 24 hours, and it is important to remember that the physical examination of the chest at this stage may be negative. Exertion during this period will bring on and aggravate the condition; absolute rest is therefore essential. Usually symptoms develop gradually, the animal becoming uncomfortable, coughing frequently with the production of a bloody, frothy nasal exudate. The pulse is at first full but becomes rapid, thin, and later difficult to detect. Dyspnoea increases as pulmonary edema develops. Respirations become hurried and labored. Little change occurs in the conjunctiva, but the nasal mucosa is at first injected, and as dyspnoea develops it takes on a bluish tinge.

(4) *Diagnosis.*—Diagnosis is made from the history of exposure and onset of symptoms some hours after exposure. Whether or not an efficient horse gas mask has been worn must be considered in the differential diagnosis.

(5) *Immediate treatment.*—Immediate treatment consists of—

(*a*) *Rest.*—Excessive movement or the lightest work will aggravate the condition. Treatment should be carried out locally so as to avoid the delay and fatigue of evacuation.

(*b*) *Heat.*—Heat in the form of warmth or blankets should be supplied so as to diminish oxygen requirements.

(*c*) *Stimulants.*—Caffein sodium benzoate might be given during the critical stage. Oxygen therapy for animals is not practicable under field conditions.

(*d*) *Venesection.*—Early and drastic blood letting should be practiced, 6 to 8 pints being taken once in 24 hours. It should be carried out as soon as the signs of pulmonary edema are established. Bleeding is contra-indicated in the later stages. When the blood is concentrated (thick) and the hemoglobin content high a less amount should be taken. Venesection reduces the intravascular pressure, relieves the heart, and accelerates the process by which the blood plasma in the alveoli is returned to the circulation.

(6) *Useless procedures.*—Useless procedures are the infusion of normal saline, injections of atropine, and especially contra-indicated is the administration of adrenalin which might further embarrass the circulation. Contra-indication of morphine for restless animals is based upon experience of the first World War. Guarded doses of barbiturates and even chloral hydrate may be tried.

(7) *Prognosis.*—If the patient survives 4 days, recovery may be expected, but the sequel of broncho-pneumonia should not be overlooked. Convalescence should not be hurried and the animal should be slowly brought back to work.

c. Chlorpicrin (PS).—(1) *General.*—Chlorpicrin is a slightly oily, colorless to brownish liquid. In the summer it is persistent for 1 hour in the open, 4 hours in the woods; longer in winter. It is a harassing and casualty agent, and has a sweetish flypaper odor.

(2) *Pathology.*—Chlorpicrin not only irritates the alveoli, but also the upper bronchi and trachea. Pulmonary edema may appear. Because of the involvement of the upper bronchi, plugging and consequent pulmonary emphysema may develop. Nephritis may also be produced.

(3) *Symptoms.*—The clinical picture is similar to that of phosgene.

(4) *Diagnosis.*—Diagnosis is made from the history of exposure and from any symptoms in men (exposed with the animals) referable to irritation of eyes, nose, and lungs, and sometimes associated with nausea and vomiting.

(5) *Prevention, protection, and immediate treatment.*—These are the same as for phosgene.

d. Chlorine (Cl).—Chlorine is a heavy, greenish-yellow nonpersistent gas with a pungent characteristic odor. In heavy concentrations it attacks the upper respiratory system, immediately causing bronchial spasm and choking cough. If death is not immediate from the intense reflex bronchial spasm, the later symptoms are similar to those of phosgene. Considerations for the protection of animals are the same as those for phosgene.

44. Vesicants.—*a. General.*—The terms "blister" and "vesicant" agents are misnomers in the case of animals. Vesication of the skin does not ordinarily occur. While the vesicants will irritate any part of the body with which they come in contact, they also function as lung irritants. In general there are two forms of vesicant agents, that is, those which cause local surface irritation, and those which cause local surface irritation plus internal poisoning. These latter compounds usually contain arsenic. More than any other type of agent, the vesicants, especially the arsenicals, will contaminate food and water, and render other supplies dangerous to handle until they have been decontaminated. Before treating vesicant agent casualties, veterinary personnel must apply to themselves those measures of individual and collective protection which are necessary to prevent themselves from becoming casualties from contact with the contaminated animals or matériel. The primary vesicant agents used in warfare include mustard (HS), lewisite (M1), and ethyldichlorarsine (ED).

b. Mustard.—(1) *General.*—Mustard is a heavy, oily, almost colorless liquid when pure, but the crude or plant-made product is dark colored. Mustard is only slightly soluble in water, but soluble in many of the organic solvents such as gasoline, kerosene, acetone, carbon tetrachloride, and alcohol; it is also freely soluble in animal fats and oils. It is slowly absorbed by rubber gloves and clothing and thus contaminates these articles. In general, efficient destruction of mustard gas is accomplished only by strong chemicals. It is persistent in the summer for from 4 to 5 days in open, for 1 week in woods; in the winter, for several weeks both in open and in woods. The period in which it is dangerous for unprotected animals to *cross* a mustard contaminated area is much less than that indicated above. Mustard has an odor of garlic or horse radish. It is emphasized, however, that prolonged exposure to even a concentration *just* detectable by odor will produce casualties in man.

(2) *Injury caused by liquid mustard.*—(*a*) The greatest amount of severe incapacitating injury to animals will be produced from contamination with liquid droplets of mustard, by spray from aircraft, by clouds of fine droplets or gross splashing from bomb and shell bursts, or by traversing ground recently contaminated with liquid. Exposure to vapors of mustard generally produces far less injury to animals than to man. Liquid mustard contamination of the horse's skin is followed in 10 to 20 minutes by an erection of the hair coat in the vicinity of the direct contamination. This persists for some time and is followed by edema, serous exudation, and in some cases necrosis with subsequent ulceration. The latter is seen more frequently when liquid contamination occurs, affecting especially areas of the skin partially or wholly devoid of hair. A full natural coat of hair will not prevent a lesion but tends to hold up penetration and to cause lateral spread. The result is a shallower lesion with a shorter healing time than would be the case if the hair had been clipped. When horses traverse ground recently contaminated and glistening areas of liquid mustard can be seen upon the foliage, the exposure results in an edematous filling around the pastern and fetlock regions. This edema and further changes lead to an intense lameness. The fine skin at the hollow of the heel is particularly susceptible and sepsis may be expected. The horny hoof, including the frog, is sufficiently resistant as to be relatively unaffected.

(*b*) The eye of the horse seems to be less susceptible to mustard vapor injury than that of man but the smallest drop of liquid into the eye causes serious injury with an acute conjunctivitis, keratitis, and temporary blindness. Necrosis of the cornea leads to opacity and blindness. There may be a mucopurulent discharge.

(*c*) The respiratory tract may be less susceptible to vapor injuries; but, in general, extensive exposures of horses to mustard vapors and mists will lead to the same type injury as is seen in man.

(*d*) Consumption of contaminated water or forage will, in addition to producing ulceration of the lips and mouth, produce a gastroenteritis accompanied by colic and blood-stained diarrhea.

(3) *Prophylaxis after contact.*—(*a*) Because the hair coat tends to hold up penetration, prophylactic measures are effective after a longer time has elapsed than would be the case with man. Immediate prophylaxis in the field is seldom possible; this procedure, however, is almost wholly successful. The healing time is shortened in proportion to the speed with which decontamination is employed. Some mitigation of the injury may be expected when the delay amounts to an hour or more.

(*b*) Liberal scrubbing of a 5-percent solution of potassium permanganate onto contaminated areas of the horse's skin appears to be a practicable field procedure for the neutralization of mustard. A 3-percent to 10-percent hydrogen peroxide solution, preferably the stronger, is also effective, but the problem of supply and storage is more difficult. Scrubbing affected areas with soap and water will remove any uncombined agent. Bleach paste (1 part chloride of lime in 3 parts cold water) is highly irritant to the horse's skin and must be washed off within 5 minutes after application, but it may be used to neutralize known areas of mustard contamination. Protective ointment may be rubbed into the hair coat for decontaminating small areas in an emergency, but it must be remembered that it is highly irritating and must be removed shortly after application or a severe dermatitis will develop. It is not believed that the removal of mustard from the horse's skin by use of such mustard solvents as gasoline, kerosene, carbon tetrachloride, or alcohol is a practicable field procedure, since this merely dilutes and does not destroy the contaminating agent. Eye lesions caused by mustard may be very severe. Affected eyes should first be freely irrigated with a 2-percent solution of soda bicarbonate, normal saline, or with water alone.

(4) *Treatment.*—Decontamination of contaminated areas of the skin should always precede treatment procedures. No specific is as yet available for mustard burns on horses and mules. Treatment will depend upon the severity of the case and the site of the lesion. The aim of aftertreatment is to counteract secondary infection. Little or no treatment is necessary in the case of slight injury to the skin that is not affected by friction of harness and saddlery. The actual treatment of mustard burns should be similar to that applied to a heat burn of like size and intensity. Cod liver oil, tannic acid, gentian violet, and like preparations have all been used with varying degrees of success. Amyl salicylate has been used successfully in the treatment of mustard injuries in man; however, this in itself produces *blistering* and *vesication* when applied to clipped areas of the horse's skin. Irrigation of injured eyes with sodium bicarbonate solutions should be employed. When the swelling and pain prevents irrigation, bathing with boric acid solution may help. Pain may be allayed by local anesthetics. Treatment should progress with the view of allaying secondary infection. Treatment of respiratory and alimentary lesions should follow general symptomatic practices. Fresh air, warmth, and nursing are essential.

c. Lewisite (M1).—(1) *General.*—Lewisite is a colorless or slightly yellow liquid, which becomes dark brown on standing. Un-

like mustard it remains effective in cold weather. Lewisite is soluble in absolute alcohol, benzine, kerosene, olive oil, liquid petrolatum, etc. It is poorly soluble in water but hydrolyzes readily to form an oxide which vaporizes very slowly but retains vesicant and systemic poisonous properties. Liquid lewisite penetrates rubber and fabrics, making it dangerous to wear rubber gloves or clothing previously contaminated. Lewisite generally is less persistent than mustard. Its freezing point is lower than that of mustard and therefore it can be used effectively in colder weather. During damp weather and when in contact with moisture it rapidly hydrolyzes to form the oxide possessing vesicant properties. This oxide may remain in contaminated soil for a long time and produce injury when coming into direct contact with the skin. It is a casualty-producing agent and the vapor has a geranium-like odor.

(2) *Injury caused by lewisite.*—Lewisite has a more rapid and severe action on the skin than mustard. In addition there may be signs of systemic effects. Skin burns resemble those caused by mustard except that they are immediately irritant and lead to considerable restlessness. In man the blister formed is filled with fluid which is also vesicant. There may be signs of various toxic changes in the viscera and arsenic may be found in all tissues. Inhalation produces ulceration of the upper respiratory tract and secondary bronchopneumonia may follow. Symptoms develop earlier and are more severe than with mustard gas. In the case of lewisite, the hair coat also raises at point of skin contamination.

(3) *Prophylactic and aftertreatment.*—Prophylactic measures for the destruction or removal of this agent must be initiated at the first opportunity, if satisfactory results are to be expected. Scrubbing the contaminated skin with a 5-percent solution of potassium permanganate, or a 3- to 10-percent solution of hydrogen peroxide, will reduce the amount of injury. This will lessen the risk of death from arsenic absorption, even if delayed approximately 30 minutes. Washing or scrubbing with soap and water is also somewhat beneficial in the removal of the contaminating agent. Aftertreatment is purely symptomatic and should follow the same lines as those used for mustard injury.

d. Ethyldichlorarsine (ED).—(1) *General.*—This compound is a liquid which is colorless when freshly prepared, but turns yellow on aging. It possesses a biting, irritant odor. It is readily soluble in alcohol, ether, benzene, and acetone. It hydrolyzes very slowly, forming a poisonous oxide. It is less persistent than lewisite or mustard but can be used either in cold or damp weather.

(2) *Injury caused by ethyldichlorarsine.*—Injury to the skin is less marked than with lewisite and mustard. In man there is an immediate irritation to the nose and throat accompanied by sneezing, lacrimation, nausea, and emesis.

(3) *Treatment, prophylactic and after.*—In general the treatment of ED casualties would follow that used for injuries from lewisite.

45. Lacrimators.—The primary lacrimators are the solid substances, chloroacetophenone (CN), and brombenzylcyanide (CA). Solutions of chloroacetophenone, dispersed as a spray, may be used. Serious irritant effects will result when the liquid spray gets into the eyes of men or animals. The solids may be dispersed from a burning candle while the solutions may be sprayed from airplanes. In very low concentrations these agents are intolerable to man, but under ordinary field conditions they have little effect upon horses and mules. Irrigation of the eyes with sodium bicarbonate will limit the irritant effects.

46. Irritant smokes.—The irritant smokes, diphenylaminechlorarsine (DM, Adamsite), and diphenylchlorarsine (DA), are sensory irritants and, when used, produce in man a severe pain in the sinuses, bones of the lower jaw, throat, and chest. Mental depression may be marked. The gas mask furnishes complete protection for man. These irritant smokes are dispersed with the aid of heat in clouds or smokes containing very fine particles. These smokes are not used for screening purposes. They may affect horses mildly but are not incapacitating. These agents are not considered persistent. Food and forage may become contaminated with DA or DM. When contamination is heavy, such supplies should not be used until it is determined by qualified personnel that they are safe for human or animal consumption.

47. Screening smokes.—*a. General.*—These agents comprise white phosphorus (WP), titanium tetrachloride (FM), sulphur trioxide-chlorsulfonic acid solution (FS), and HC mixture (HC). They are used to screen position or troop movements or to mask gas cloud attacks by other agents. In general the smoke from these agents is not toxic in the ordinary field concentrations, but liquid FS and burning WP may be dangerous at the immediate site of dispersion. Because of their nature these materials are nonpersistent. WP and FM give white smoke clouds, HC gray. The smoke from FM, HC, and WP is not ordinarily irritating, whereas that from FS is corrosive to metals and may in some cases be irritant to the eyes, skin, and respiratory tract. WP is dispersed by explosives, HC by candles only, FS and FM from spray or from explosives.

b. White phosphorus (WP).—(1) *General.*—This is a pale, yellow, wax-like solid which bursts into flame when exposed to the air, and must be stored under water or oil. Incendiary properties are enhanced when WP filled shells burst, scattering burning particles over an area. WP has an odor of burning matches.

(2) *Pathology.*—Particles of white phosphorus produce deep burns on the skin with which they come into contact. The smoke is nontoxic, but excessive long exposure to the *fumes* of WP may cause atrophy of the liver, osteitis, and bone necrosis.

(3) *Treatment.*—Smother the burn immediately with water, mud, or other air-impervious materials. Keep the burn under water until a 2- to 10-percent copper sulfate solution can be applied, which excludes air by forming a metallic coating on the particles. The particles can then be removed with forceps or hemostats. Further treatment is that for ordinary heat burns.

c. Sulfur trioxide-chlorsulfonic acid solution (FS).—(1) *General.*—This agent is a fuming, highly corrosive liquid which upon contact with moisture forms hydrochloric and sulphuric acid. FS has an acrid odor.

(2) *Pathology.*—Acid burns of the skin or mucous membranes are produced by contact with the liquid. It is not believed that this agent will severely affect the horse's skin.

(3) *Treatment.*—Eye burns are most important and irrigation treatment should begin as soon as practicable following exposure to irritating concentrations. A 2-percent sodium bicarbonate solution may be used for eye irrigations. This is also used for skin burns on man.

d. Titanium tetrachloride (FM).—(1) *General.*—This agent is a liquid which hydrolyzes rapidly in contact with the moisture of air to form titanium hydroxide and hydrochloric acid. The smoke has an acrid odor. It may produce acidlike burns on the skin and in the eyes but is not as irritant or corrosive as FS. Treatment of injuries is the same as for FS.

e. HC mixture (HC).—This agent is made up of a mixture of several agents. The smoke itself has an acrid smell and is composed essentially of zinc chloride and free carbon. Under field conditions this smoke is not irritating to man or animal. No treatment is needed.

48. Incendiary agents.—These agents are used primarily to cause fires in matériel, but if they come into contact with the body will produce severe burns. White phosphorus in an explosive shell or aerial bomb is also an incendiary agent, as has been previously

pointed out. Treatment of injuries from any of the incendiary agents follows the general procedures used for any heat burn.

49. Systemic poisons.—*a. General.*—In addition to the agents considered previously under the physiological classification as lung irritants, vesicants, lacrimators, and irritant smokes, there are other materials which may be used directly or indirectly to produce casualties, and which may be conveniently designated "systemic poisons." In general they produce their main effects only after absorption into the blood stream. The most important of these agents are hydrocyanic acid, arsine (arseniuretted hydrogen), and hydrogen sulfide. Protection against these agents will require an efficient canister type of horse gas mask.

b. Hydrocyanic acid.—(1) *General.*—This agent is a colorless, highly volatile, nonpersistent agent, and its vapor rapidly disperses in air. It has an odor of bitter almonds or peach kernels. Hydrocyanic acid produces asphyxia by hindering the oxidative processes of the tissues, and depresses and paralyzes the central nervous system, beginning with the medulla. Its casualty-producing action on animals is either all or none. If it does not kill rapidly it probably will not produce many treatable casualties. Efficacy of treatment in borderline cases is limited by the extremely rapid course of the poisoning. Animal therapeutic measures in war therefore do not seem practicable. The inhalation of amyl nitrite fumes and intravenous injections of sodium thiosulfate have been reported as useful in the treatment of borderline cases in man.

c. Arsine.—(1) *General.*—This is a colorless, nonpersistent, casualty-producing, inflammable gas. Its vapor has a garlic-like odor and metallic taste; however, its inhalation in concentrations not detectable or recognizable by odor may cause toxic symptoms.

(2) *Symptoms.*—Arsine produces no irritation of the skin but when it is inhaled and absorbed the red blood cells are hemolyzed. The kidneys may be blocked by various degrees of the debris from red blood cell destruction; anuria and uremia may result. Hepatitis is a common complication. A few hours after exposure to high concentrations, patchy sweating, increased respiration, anuresis, and an increased temperature may be noted. These symptoms may be accompanied or followed by anemia. Severe hemoglobinuria may appear early.

(3) *Treatment.*—The essentials of treating arsine poisoning in animals consist of inducing diuresis, thereby decreasing kidney blockage. This may be accomplished by allowing the animal free access to water and rendering the urine alkaline by large doses of sodium bicarbonate.

Anemia may be treated by the usual methods. The horse gas mask gives protection against this agent.

d. Hydrogen sulfide.—This is a colorless, nonpersistent gas, heavier than air, with a characteristic odor of rotten eggs. It is detectable at harmless concentrations. Following exposure to high concentrations there may be local irritation to the eyes and respiratory tract, and pulmonary edema may result. Treatment is same as for phosgene.

50. Incidental gases.—These include certain gaseous compounds, such as carbon monoxide, nitric fumes, and ammonia. They may be met with indirectly in military operations as the result of fires or shell explosions in confined or poorly ventilated spaces. Carbon monoxide, and to a lesser extent nitric fumes, are combustion products which may remain for some time in freshly formed shell holes. Ammonia may be liberated in refrigeration plants, accidentally or as a result of enemy bombing activities. The horse gas mask canister furnishes protection to horses against nitric fumes but not against ammonia or carbon monoxide. Nitric fumes or those of ammonia may produce pulmonary edema.

51. General consideration of animal casualties.—*a.* The prevention of gas casualties in animals is of utmost importance in eliminating lost working days, both to the exposed animals and of the attending personnel. This can be greatly facilitated by the proper use of protective equipment and of accepted principles of protection.

b. The use of individual protective devices will aid greatly in the prevention of chemical warfare casualties among horses; horse gas masks, protective leggings, and individual covers are probably of most importance.

c. Collective principles of protection are also effective in eliminating casualties. Stables, veterinary hospitals, aid stations, picket lines, corrals, and bivouac areas should be placed a sufficient distance from the front lines to eliminate, so far as possible, exposure to gas attacks, care being taken to avoid small valleys and ravines where high concentrations of gases are likely to occur. The proximity of roads and strategic points is hazardous and should be avoided, since such localities are likely targets for gas and high explosive shells. Animals should be prevented from drinking from water holes, trenches, or shell craters, and from pasturing in areas which have recently been contaminated until the forage and water in such localities is determined suitable for animal consumption. Treatment of contaminated water to render it potable is not feasible except by water supply personnel of the division, corps, or army engineer units. Water in deep

wells or large running streams and lakes, where the factor of dilution is great, can usually be considered to be nontoxic.

d. The comparative physiological effect of exposing horses and men to casualty-producing chemical warfare agents appears to be similar; however, the problem of providing protection against them may differ. When used, the horse gas mask provides protection comparable to that furnished man by the service gas mask. In combat, however, vesicant protective equipment must provide the foot soldier complete protection for his body, since he is more or less fixed to a locality and when under fire is probably in close contact with the ground, even lying in foxholes for his protection. Due to mobility, the horse may be expected in some cases to avoid contaminated terrain, thus eliminating respiratory and vesicant injury. In sudden attacks or under certain tactical conditions, animals may be forced through liquid vesicant contaminated terrain where severe skin injury conceivably will result, especially in instances where liquid vesicant contamination of the lower leg may occur from direct contact with contaminated brush and grass. When only vapors of vesicants are encountered, horses may receive considerable exposures without developing incapacitating injuries, especially when protected with the gas mask.

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BY ORDER OF THE SECRETARY OF WAR:

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The Adjutant General.

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TREATMENT OF CASUALTIES FROM CHEMICAL AGENTS

Prepared under direction of The Surgeon General

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SECTION I

GENERAL

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1. Object.—The object of this manual is to supply information and provide instructions for military medical personnel relative to the care and treatment of casualties resulting from exposure to chemical agents. This manual is supplemental to FM 21–40.

2. Chemical casualties.—*a.* Chemical casualties may occur not only as the result of direct exposure to chemical attack, but also indirectly from the consumption of chemically contaminated food and water or contact with similarly affected material.

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b. Chemical agents are generally employed in amounts sufficiently great to produce high concentrations on the target, and in the event of such attacks medical officers should be prepared to handle proportionately larger numbers of casualties than those which occur in other forms of combat.

3. Fundamentals of treatment.—*a.* Prophylactic measures are of great importance in reducing the incidence of potential chemical casualties, as immediate treatment following exposure is usually much more beneficial than therapeutic measures applied after symptoms of injury have begun to appear. Since the type of preventive treatment is often specific, some knowledge of the properties of chemical agents is desirable, if not essential, for the effective utilization of preventive measures.

b. The purely symptomatic treatment applied after injury has been produced is usually quite different from the specific prophylactic treatment, and usually does little to prevent its further damage. In general, the definitive treatment of injuries caused by chemical agents does not differ from that of similar conditions occurring from other causes, i. e., the after treatment of a skin lesion caused by mustard is the same as that for any ulcerative lesion of the skin of like degree.

Section II

LUNG IRRITANTS

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4. General.—*a.* Under some conditions practically all of the chemical agents are lung irritants. With certain of these, however, under concentrations encountered in the field, this power to produce lung irritation is their main characteristic. These primary pulmonary irritants are phosgene, chlorpicrin, and chlorine.

b. With all lung irritants a latent period of from 1 to 12 or more hours may intervene between exposure and the onset of symptoms, objective or subjective. It occurs regularly after exposure to phosgene and is apt to be longer than 12 hours, but it may be absent or very short after exposure to high concentrations of chlorine. Because of this latent period it may be difficult at times to determine whether exposure has actually occurred. Common knowledge of the existence of the latent period may be a source of malingering. The fact that the smoke from a cigarette is unpalatable after inhalation of lung irritants, even

during the latent period, may be of help in detecting such malingering. However, suspected cases must be given the benefit of any doubt and kept under observation and at rest for 24 hours.

c. The most important and the most serious effect of pulmonary irritation is the occurrence of pulmonary edema, which may be caused by any of the agents listed above. This condition is produced essentially by irritation of the walls of the bronchioles, alveoli, and the adjacent capillary endothelium, and results in a tremendous outpouring of fluid into the alveoli. Clinically this is manifested by decreased expansibility of the lungs, increases in density which may not be revealed by changes in the percussion note or by the transmission of breath or voice sounds, and also by the presence of moisture indicated by rales of varying types. Physiologically, this pulmonary edema interferes very seriously with the gaseous exchange in the lung and so contributes to the development of an anoxemia. Moreover the edema plus the local changes in the blood vessel wall, including thrombosis and the increased viscosity of the blood, all interfere with the circulation through the lung and add to the anoxemia. If this interference with the circulation is maintained, general cardiac collapse may ensue.

d. Other gases, such as nitric fumes, etc., are also pulmonary irritants, and require the same preventive and treatment measures as are necessitated by the typical chemical lung irritants.

e. Adequate protection against field concentrations of the lung irritant gases used in warfare is provided by the service gas mask and collective protectors.

5. Phosgene (CG).—*a. Pathology.*—Relatively high concentrations of phosgene may be breathed without immediate irritation of the respiratory tract, hence the most distant bronchioles and alveoli of the lung are exposed to the gas. In contact with the moisture of the alveolar walls, the gas hydrolyzes, producing hydrochloric acid and carbon dioxide: $COCl_2 + H_2O \leqslant 2\ HCl + CO_2$. The irritant effect is supposed, but not proved, to be due to the action of the hydrochloric acid thus formed. As a result of this irritation the permeability of the alveolar and capillary walls is increased, causing a pulmonary edema. Pulmonary vessel thrombosis may develop. There are relatively few changes in the upper respiratory tract.

b. Symptoms.—Actually there may be no respiratory symptoms of any sort until pulmonary edema develops suddenly and dramatically. Sometimes this latent period may last for as long as 12 to 24 hours. This insidious period, during which no symptoms develop, may be very deceptive because the individual may not realize that he has

been gassed, and thus he may not receive early enough the absolute rest which he requires. Moreover, it is important to remember that the physical examination of the chest at this stage may be entirely negative. Sometimes there is a gradual development of symptoms with redness of the eyes, flushing of the face, increased respiration and occasionally a painful cough, although the cough is not common. Even with this clinical form, the results of the physical examination of the chest may still be negative. In more severe cases of phosgene poisoning, whether or not the insidious latent period occurs, the patient may develop flushing of the face and coughing followed rapidly by a deep cyanosis with engorgement of the neck veins (the blue type of asphyxia). He then becomes very uncomfortable, coughing frequently with the production of bloody, frothy sputum; from the bluish, mottled appearance of his face he appears to be slowly strangling. Even with the pulmonary edema and failure of the right side of the heart, the chest examination may or may not show changes in the percussion note; the breath sounds and voice sounds are not intense; fine rales may be heard over the back and sides and in axillae, and rough rhonchi over the upper chest. During this stage the patient may suddenly or gradually develop cardiac failure manifested by a grayish leaden hue of the skin, collapse of the neck veins, and cold, clammy skin (gray type of asphyxia); in general he may simulate a shock case, his color, however, appearing more greenish gray than in the usual shock patient. In this gray type of asphyxia, the chest examination may yield the same findings as occur with the blue type of asphyxia. The blood pressure, however, is lower and the heart sounds at times are much less intense. Instead of passing through the usual course from the blue to the gray type, the patient may suddenly go into the gray stage immediately after the insidious latent period. He may die during the gray period or some days afterward with a secondary broncho-pneumonia. If recovery occurs from the acute phase, he will be weak and dyspneic and because of his exhausted condition absolute rest must be continued.

c. Diagnosis.—Diagnosis is made from the history of exposure or the sudden onset of symptoms occurring some hours after exposure.

d. Immediate treatment.—(1) This consists of absolute rest, application of heat (blankets, warmth, hot drinks), inhalation of oxygen, and venesection in the "blue" cases. Even though rest appears unnecessary it must be enforced, the patient being kept in a prone position on a litter, the foot of which may be elevated to assist lung drainage. He should be kept well covered with blankets and given hot drinks of coffee, tea, or water; alcoholic drinks should not be used.

Even if the patient is very restless and apprehensive, morphine should never be given. Contraindication of morphine is based on the uniform experience of the World War. Restlessness and apprehension may be combated with guarded doses of the barbiturates. However, the relief afforded by oxygen inhalation is the best means of quieting and reassuring the patient.

(2) *Oxygen therapy.*—Once pulmonary edema has developed, there is serious interference with respiratory exchange causing anoxemia, which should be relieved by the administration of oxygen. Mixture of helium with the oxygen offers no advantage, since the difficulty is not bronchial obstruction but the filling of alveoli with fluid. Mixture of carbon dioxide with the oxygen is likewise not indicated, since there is no decrease in the carbon dioxide of the blood. Furthermore, under field conditions, the weight of oxygen cylinders is a serious problem for transportation. It is therefore better to have the weight which must be transported represent pure oxygen rather than oxygen diluted with any other gas. Oxygen administration should be started as soon as pulmonary edema develops. This can best be accomplished by means of an efficient nasal mask which insures an adequate yet economical supply of oxygen to the patient. With such a mask, oxygen administration for 3 or 4 minutes out of every 15 may suffice, provided this treatment is continued night and day as long as cyanosis tends to recur, the relief of this condition being the best indication by which to regulate the supply of oxygen. When such a device is not available, oxygen may be given by other methods, such as the insertion of a nasal catheter, or the much less efficient funnel technic. If the military situation limits the supply of oxygen it should be remembered that oxygen therapy is most useful in cases that verge on the desperate, especially those which are still deeply cyanotic on the second day.

(3) *Venesection.*—Because it can easily be performed under field conditions, bleeding is probably the most valuable therapy for the cyanotic or blue stage of phosgene poisoning, but *it should never be employed in the gray or collapsed stage;* 400 to 600 c. c. of blood should be withdrawn as early as possible and another 500 c. c. may be removed 6 hours later and the procedure repeated if necessary. A large bore needle should be used unless the viscosity of the blood makes direct incision of the vein necessary. Cardiac stimulants such as digitalis, caffeine, and coramine have been found of little or no value in combating the cardiac failure. Oxygen inhalation, by relieving the anoxemia under which the heart muscle is laboring, and venesec-

tion, by relieving the load on the heart, are more useful than cardiac stimulation.

e. Further treatment.—Absolute rest must be continued for some time after the acute respiratory symptoms and signs disappear. Resumption of exercise should be undertaken very gradually, care being taken to avoid fatigue or exhaustion. At first, toilet privileges may be granted, then the patient may be allowed to sit up for brief intervals in a chair. Alternate walking and resting for short periods is the next step. Later the convalescent should march short distances and the point at which dyspnoea develops determined. The possibility of malingering should be considered and ruled out by objective symptoms. If broncho-pneumonia develops, which is seldom, it is more apt to occur in the first few days than in the convalescent period and may be treated as broncho-pneumonia from any other cause. Sulfonamide therapy may be used. Oxygen inhalation may be resumed whenever cyanosis with dyspnoea occurs. If anesthesia is required for surgical procedure, local, nerve block, or spinal administration should be utilized, if possible. When it is necessary to employ inhalation, anesthesia, chloroform, or nitrous oxide should be used.

f. Prognosis.—Prognosis should always be guarded because of the insidious nature of the poisoning. The milder cases always recover. Most deaths occur within the first 24 hours. The prognosis is worse in the pallid or gray than in the blue cases. Deaths taking place after some days are largely due to broncho-pneumonia, which is more apt to occur in men with pre-existing pulmonary disease. Only occasionally are chronic pulmonary diseases, such as bronchitis and bronchiectasis, caused by phosgene poisoning; pulmonary tuberculosis never. Psychosomatic (pulmonary) complaints may continue for years.

6. Chlorpicrin (PS).—*a. Pathology.*—Chlorpicrin irritates not only the alveoli but also the upper bronchi and the trachea. Pulmonary edema with all of its concomitants may appear, similar to that produced by phosgene. The pulmonary vessel thromboses, however, are not as common as with phosgene. Because of the involvement of the upper bronchi, plugging and consequent pulmonary emphysema may develop. Nephritis may also be produced.

b. Symptoms.—The clinical picture is similar to that of the various phases of phosgene poisoning. In addition, nausea and vomiting are much more marked with chlorpicrin than with phosgene poisoning.

c. Diagnosis.—The cardinal points are history of exposure; symptoms referable to the eyes, nose, and lungs; nausea and vomiting; characteristic flypaper odor on clothing.

d. Treatment.—The therapy is the same in all respects as for phosgene. If hot drinks aggravate the nausea and vomiting they should be withheld temporarily.

e. Prognosis.—The prognosis is worse when pulmonary edema develops. Sudden deaths are very rare. Emphysema and chronic bronchitis may follow acute chlorpicrin poisoning.

7. Chlorine (Cl).—*a. Pathology.*—There is marked irritation of the upper respiratory tract, trachea, and bronchi, which may progress to extensive necrosis of the mucous membrane of the trachea and bronchi. With equal exposure, chlorine produces less alveolar damage than phosgene, probably because of its greater solubility in the moisture of the upper air passages and spasmodic closure of the upper air passages preventing the gas reaching the alveoli. Alveolar damage, with pulmonary edema, when it occurs, is similar to that caused by phosgene.

b. Symptoms.—Unlike phosgene, chlorine is violently irritating to the upper respiratory tract. Rarely, sudden death may occur after the first inhalation of a very heavy concentration due to reflex action. After exposure to the lower concentrations usually encountered in the field, the first symptoms are a burning sensation in the throat, violent paroxysmal coughing, and a feeling of suffocation. The signs and symptoms of pulmonary edema then supervene, usually after a shorter time than in phosgene poisoning, sometimes as soon as 20 minutes after exposure. The picture in this stage is like that of phosgene poisoning, added to which is a choking cough. If the pulmonary edema subsides, bronchitis may persist for several weeks.

c. Diagnosis.—The cardinal points are history of exposure, odor on clothing, intense irritation of upper respiratory tract with paroxysmal cough, and persistent bronchitis during convalescence.

d. Treatment.—This is the same in all respects as that for phosgene.

e. Prognosis.—The prognosis is poor when pulmonary edema has been established. Chronic bronchitis may be persistent. Some pulmonary emphysema may be permanent. Most patients surviving pulmonary edema ultimately make a complete recovery.

SECTION III

VESICANTS

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8. General.—*a.* While the vesicants are employed primarily for their action on the skin, they irritate any part of the body with which they come in contact, and thus function also as lung irritants. Conversely, some other agents not primarily employed to irritate the skin may nevertheless function in this manner, as for example certain of the irritant and screening smokes and lacrimators. In general, there are two forms of blistering agents, i. e., those which cause only local surface irritation and those which cause local surface irritation plus internal poisoning. These latter compounds usually contain arsenic.

b. More than any other type of chemical agents, the vesicants, especially the arsenicals, will poison food and water, and render other supplies dangerous to handle until they have been decontaminated.

c. Before treating vesicant agent casualties, medical personnel must apply to themselves those measures, individual or collective, which are necessary for their own protection, otherwise they may themselves become casualties.

d. As a rule, information concerning the type of causative agent used will be furnished the medical officer in advance of the stream of casualties by the military intelligence officer, and additional knowledge may be obtained from the patients themselves. In turn, medical officers should notify the military intelligence officer as to what types of agents are responsible for the casualties which are being received.

e. The service gas mask protects only the face, eyes, and lungs against vesicants. Protective clothing designed for protection of the body against gases of the mustard type will be issued in time of war, and protective ointment will be issued for application to insufficiently protected areas such as the wrists, hands, and neck. This ointment will furnish complete protection against high vapor concentrations, and significant protection against droplets of mustard when mixed with the latter on the skin by rubbing.

9. Mustard (HS).—*a. Pathology.*—There is marked destruction of the skin, in most instances involving the lower portion of the dermis, and the picture resembles that of an X-ray burn. Marked irritation of the upper respiratory tract occurs with ulceration of the trachea and bronchi, commonly followed by secondary infection and the development of broncho-pneumonia. It is this broncho-pneumonia which is responsible for most of the deaths.

b. Symptoms.—Signs and symptoms following exposure to mustard gas are delayed, the length of the latent period depending on the concentration of the agent and also on the individual sensitivity of the skin. Prolonged exposure to such low concentrations as are just detectable by odor will produce casualties. In personnel unprotected

by masks, eye symptoms are generally the first to appear. These begin 2 or 3 hours after exposure to vapor, at first consisting of smarting and watering, then proceeding to reddening, pain, edema, and the phenomena of acute conjunctivitis. Sneezing and increased nasal secretion are also early symptoms. Skin burns from vapor may not appear until 12 or more hours have elapsed, but may occur within 1 hour after contact with liquid mustard. The first symptom may be severe itching of the skin, soon followed by a sunburn-like erythema, upon which small and large blisters develop. Shortly before the development of visible blisters, the surface of the reddened skin can be rubbed raw with slight pressure and friction. In lesions from liquid splashes, the blisters may be arranged in a peripheral ring about a central, whitish, indurated area. The blisters are surrounded by a zone of erythema. Because of the depth of skin destruction, mustard lesions require some weeks to heal, and secondary infection may occur. There is little systemic reaction except where there are extensive burns or marked secondary infection. Skin hypersensitivity to mustard may follow repeated exposure. Vapor burns are more severe on those areas of the body covered by clothing, which interferes with dissipation of the mustard, and also on those areas which are subject to friction, or where the skin is moist or thin. These more easily injured areas include the perineum, external genitalia, axillae, elbows, knees, and neck. Very deep burns of the skin may be produced by splashes of liquid mustard. The fluid contents of mustard blisters are neither vesicant nor irritating when placed on normal skin. Eye burns vary, according to exposure, from simple conjunctivitis with lacrimation and photophobia to severe corneal ulceration. Irritation of the upper respiratory tract is shown by hoarseness, then possibly cough, and later muco-purulent sputa. Broncho-pneumonia is an ordinary complication of the pulmonary irritation. Gastrointestinal irritation of varying degrees may follow the ingestion of mustard.

c. Diagnosis.—The essential factors upon which diagnosis is based are as follows:

(1) History of exposure.

(2) Mustard or garlic odor on skin and clothing.

(3) Itching of skin with little or no pain during the development of lesions.

(4) Irritation and reddening of conjunctiva and eyes.

(5) Erythema of skin followed by blistering, which develops only some time after exposure.

(6) Blisters surrounded by zone of erythema (differential diagnosis from lewisite).

d. Prophylaxis.—To be effective, prophylactic measures must be instituted within a few minutes; immediate prophylaxis is effective following liquid contamination, but of little use after exposure to vapor, since in this case practically all of the agent has already penetrated the skin. Contaminated clothes must be quickly removed, using the proper precautions. A very careful technic must be followed for the removal of mustard from the skin, otherwise such attempts merely serve to spread the agent. First, dry pads are very gently applied to absorb any mustard remaining on the skin. Next, the area is dabbed gently and repeatedly with sponges dampened with gasoline, kerosene, carbon tetrachloride, or alcohol. The skin surface within and beyond the outlines of the contaminated area is then vigorously scrubbed with soap and water and then the area is patted dry. The sponging materials contaminated in this process must be burned or buried. The protective ointment also removes mustard effectively from the skin surface if it is applied with rubbing and then wiped off. Products containing active chlorine, such as bleach and sodium hypochlorite (Dakin's solution), may also be used. Because of the irritant properties of these prophylactic materials they must be removed as soon as possible (at least within a few minutes) from the skin surface. Bleach may be used as a paste, 1 part to 1 or 2 parts of water. If water is not available, a small amount of dry bleach may be used. There may be some injury from the heat of the reaction but it will be less than that from mustard alone. If the eyes have not been protected by a gas mask, they should be irrigated with 2 percent sodium bicarbonate solution. A 2 percent butyn solution may be instilled in the eyes to relieve pain; cocaine must not be used, since, under these circumstances, it causes corneal irritation and may predispose to actual ulceration. The eyes must *not* be bandaged. If it is suspected that mustard has entered the buccal or naso-pharyngeal cavities, the mouth may be rinsed and the throat gargled repeatedly with 2 percent sodium bicarbonate solution. Prophylactic measures following the inhalation of heavy concentrations of mustard should be directed against bronchitis and the development of broncho-pneumonia.

e. Immediate treatment.—(1) With the development of erythema the skin should first be treated with solvents by the careful technic described above. If chlorinating agents are used they will aggravate the erythema. Very little else can be done for the skin during this period save to relieve the itching. This may be done with hot applications or antipruritic ointments among which antipruritic ointment No. 71 (see par. 5*e*, app.) has been found to be effective.

(2) If the eyes are involved they must be irrigated continuously for some hours with 2 percent sodium bicarbonate solutions; 2 percent butyn solution may be installed to relieve the pain. As indicated above, *no cocaine* should be employed nor should the eyes be bandaged.

(3) In early involvement of the respiratory tract, relief may be secured by gargling or spraying the throat with 2 percent sodium bicarbonate, or from benzoin and menthol inhalations.

f. Further treatment.—(1) All blisters on the skin should be opened, the fluid and dead skin removed, and the injured area blotted with sterile gauze. The tannic acid treatment ordinarily used for burns is recommended for the further treatment of these denuded areas. A freshly prepared 2 to 5 percent solution of tannic acid may be used, but an even more convenient preparation is a solution of the compound tannic acid powder (see par. 5*c*, app.) which has the advantage of keeping well in either the dry or solution form and of being a balanced saline solution. Tannic acid solution may be applied by sponging or spraying, followed after 15 minutes by 10 percent silver nitrate solution, used in the same manner, this sequence being repeated as often as necessary until a firm eschar is formed. A large sterile dressing should be applied over this crust and inspected daily. If there is any suspicion of infection under the crust that portion must be removed, otherwise it is left on for several weeks. Under certain field conditions, after the blister cover has been removed, a generous supply of the tannic acid solution may be applied on gauze and the area covered with a large sterile bandage. If there is no infection the bandage adherent to the crust need not be removed for some time. On areas such as flexor surfaces where the tannic acid treatment is not applicable, 1 percent gentian violet solution or Dakin's solution dressings may be used. Alternately, especially in burns where healing is sluggish, the denuded areas may be kept covered with gauze saturated with crude cod liver oil, or treated with cod liver oil ointment (see par. 5*h*, app.). The antipruritic ointment mentioned above may be used later for the relief of itching. If the skin lesions become infected they should be treated with Dakin's solution or other nonirritating antiseptic preparations; if available, drugs of the sulfonamide group, either in the powdered form or as 1 percent solutions, may be applied directly to the lesions.

(2) If the eyes are involved, 2 percent butyn solution may be instilled if necessary for relief of pain. Cocaine must *not* be used for this purpose. Irrigation with 2 percent sodium bicarbonate solution is continued for 24 to 48 hours after which the eyes may be very lightly covered with sponges wet with boric acid solution or normal saline.

The eyes should not be bandaged, relief from blepharospasm and photophobia being given instead by darkening the room. If the cornea is ulcerated, 0.5 percent atropine solution should be instilled to maintain dilatation of the pupils to prevent synechiae. If the eye discharge becomes purulent, colloidal silver irrigations or cod liver oil ointment should be used. In severe secondary infection nonspecific protein or heat therapy should be tried.

(3) When broncho-pneumonia occurs secondary to the pulmonary irritation, it should receive the same treatment as broncho-pneumonias from any other cause, and sulfonamide therapy should also be tried.

g. Prognosis.—In spite of widespread areas of skin involvement the prognosis is usually good. Burns from splashes require a longer time to heal than those from vapor. With secondary infection, healing may also be delayed. Occasionally, pigmentation of the skin may persist for some time after the skin lesions have healed. In spite of their appearance, vapor burns of the eye have a very good prognosis; with liquid burns the prognosis is poor, especially if the cornea is damaged severely enough to scar. Despite the large number of eye casualties in the World War, relatively few instances of permanent blindness occurred. The majority of deaths are from broncho-pneumonia secondary to pulmonary irritation.

10. Lewisite (M–l).—*a. Pathology.*—More rapid and more severe blistering and necrotic lesions of the skin are produced by lewisite than by mustard. The blister fluid is vesicant. In addition there may be signs of various toxic changes in the viscera, and arsenic may be found in all tissues. Inhalation produces ulceration of the upper respiratory tract and secondary broncho-pneumonia may follow.

b. Symptoms.—Symptoms develop earlier and are more severe than with mustard gas. Conjunctivitis, sneezing, and nasal irritation may occur. Erythema develops 15 to 30 minutes after exposure, and bullae next appear, reaching their peak in 12 hours or less. At first the contents of the lewisite blisters are more opaque than those of mustard gas. There is little, if any, erythema around the early vesicle, the entire involved area being covered by the latter. With skin burns, arsenical poisoning may occur, being manifested by dryness and soreness of the throat, diarrhea, and restlessness; later there may be signs of involvement of other structures such as those of the nervous system. Severe burns of the eyes are produced by very small amounts of the liquid. In general, lewisite acts more quickly and more painfully than does mustard gas.

c. Diagnosis.—Diagnosis is based upon the following factors:

(1) History of exposure.

(2) Odor on skin and clothing.
(3) Prompt irritation of eyes and conjunctiva.
(4) Rapid onset of skin injury with severe pain and blistering.
(5) Lack of erythema about the early vesicle.
(6) Associated severe nasal irritation.
(7) Presence of arsenic in the blister fluid.

d. Prophylaxis.—Prophylaxis must be instituted within 1 minute after exposure to be really effective against liquid lewisite. Contaminated clothing must be quickly removed with the usual precautions and, if possible, treatment should be started at the same time. The contaminated skin areas should be swabbed alternately and repeatedly with sodium hydroxide solution and with alcohol. A 10 percent solution of sodium hydroxide in 30 percent glycerine (see par. 5*b*, app.) is recommended for this purpose, but if this is not available a 5 percent solution of sodium hydroxide may be used; water may be substituted for the alcohol if the latter is not obtainable. If none of these materials are available, the same solvents and technic described for mustard (par. 9*d*) may be used. When the swabbing has been completed, the skin should be thoroughly washed with soap and water.

e. Immediate treatment.—The first procedure is to apply the prophylactic measures described above. Sometimes all that can be done is to attempt to relieve the itching by the use of simple dressings or by the application of antipruritic ointments, as recommended for mustard (par. 9*e*). When burns are produced by liquid lewisite, the most effective treatment up to 24 hours is complete excision of the burned area, provided mobility of the skin makes this procedure feasible. The excision should be carried widely about the area and down to the subcutaneous tissue, and the wound left open to heal by second intention. When this technic is not practicable, multiple scarification of the contaminated area with immediate suction is indicated. At this same time, general measures to combat the arsenical poisoning should be instituted. These include adequate fluid intake and output, intravenous glucose, high carbohydrate diet and vitamin mixtures, and the ingestion of ferric hydrate paste (see par. 5*d*, app.) mixed with water. The eyes should be irrigated with 2 percent sodium bicarbonate solution.

f. Further treatment.—The treatment of the erythema and vesicles should be continued by the local application of ferric hydrate paste (see par. 5*d*, app.). Blisters may be treated in the same manner as those caused by mustard. General as well as local treatment of the eyes must be continued. If pulmonary irritation is present, admin-

istration of the sulfonamide group of drugs should be tried as a preventive measure against the development of broncho-pneumonia, or as an adjunct to the usual treatment of this condition if it develops.

g. Prognosis.—Lewisite is much more dangerous than mustard. Fatal arsenical intoxication may result from an extensive burn, and in any event burns from liquid splashes require longer to heal than burns from vapor exposures. With liquid burns of the eyes, the prognosis is likewise serious. Broncho-pneumonia following pulmonary irritation is also a dangerous sequel.

11. Ethyldichlorarsine (ED).—*a. Pathology.*—Irritation of the nose, naso-pharynx, and lungs occurs. Vesiculation of the skin is less marked than with lewisite or mustard. Arsenical involvement of the viscera and varied post arsenical neuritides may occur. Paronychial swellings of the finger may also be present.

b. Symptoms.—There is immediate or delayed irritation of the nose; when the reaction is delayed the untrained individual may remove his gas mask, believing it to be inefficient, and will thus get a larger dose of the agent. Other symptoms are sneezing, a burning sensation in the throat, photophobia, lacrimation, nausea and vomiting, and perhaps blistering of the skin.

c. Diagnosis.—The following factors should be considered in making the diagnosis:

(1) History of exposure.
(2) Pungent odor of skin and clothing.
(3) Rapid, intense sternutatory irritant and vesicant symptoms.
(4) Disappearance of the nasal irritation within an hour.
(5) Presence of arsenic in the blister fluid.

d. Prophylaxis.—Prophylactic measures must be applied very rapidly but it may be impossible to avoid nasal irritation. Inhalation of low concentrations of chlorine from bleach powder in a wide-mouth bottle may relieve the respiratory symptoms. The skin should be dabbed with a 10 percent solution of sodium hydroxide in 30 percent glycerine (see par. 5*b*, app.), and then washed with soap and water.

e. Immediate and later treatment.—Treatment in general is the same as that described above for lewisite and mustard (pars. 9 and 10).

f. Prognosis.—There are usually no serious complications from exposure to ordinary field concentrations, but neurological disturbances and arsenical poisoning may follow exposure to very high vapor concentrations or to the liquid.

SECTION IV

LACRIMATORS

Paragraph
Symptoms and treatment 12

12. Symptoms and treatment.—*a.* The more important lacrimators are chloracetophenone (CN), chloracetophenone solutions (CNS and CNB), and brombenzyl cyanide (CA).

b. General symptoms produced by the lacrimators include lacrimation, photophobia and blepharospasm, also some irritation of the nose and the freshly shaven face. In addition, chloracetophenone solutions CNS and CNB may cause some mild papulo-vesicular dermatitis, especially in warm weather, and occasional vomiting. Direct contact of the eyes with these solutions may cause permanent damage from corneal ulceration.

c. Individuals exposed to lacrimators should be removed quickly from the contaminated atmoshpere, and faced to the wind with the eyes open. If this procedure is not possible, the mask should be put on and rapid breathing maintained to aid removal of the agent from the eyes, which are kept open as much as possible. If the eyes are markedly irritated they may be irrigated with boric acid or 2 percent sodium bicarbonate solutions. Skin irritation may be treated by sponging with 4 percent sodium sulfite in 50 percent alcohol (see par. 5*g*, app.). Under no circumstances should the eyes be rubbed. Benzol poisoning from exposure to high concentrations of the CNB solution is possible but not probable. The action of the lacrimators is prevented entirely by gas masks or by collective protection.

SECTION V

IRRITANT SMOKES (STERNUTATORS)

Paragraph
General 13
Diphenylaminechlorarsine (DM) (adamsite) 14
Diphenylchlorarsine (DA) 15

13. General.—*a.* The most important irritant smokes are diphenylaminechlorarsine (DM, adamsite) and diphenylchlorarsine (DA); they produce sensory irritation of the nose, throat, and eyes. Because of the delayed action of these agents, symptoms may not appear in slightly exposed individuals until after the gas mask has been put on. The uninformed person, believing his mask to be inefficient, may then remove it and become a casualty from further exposure to this agent or others accompanying it.

b. Adequate protection against the irritant smokes is afforded by the service gas mask and by collective protectors.

14. Diphenylaminechlorarsine (DM).—*a. Pathology.*—DM produces local irritation of the nose and nasal accessory sinuses, throat, and eyes.

b. Symptoms.—These consist of pain and a feeling of fullness in the nose and sinuses accompanied by severe headache, followed by sensations of intense burning in the throat and tightness and pain in the chest. Irritation of the eyes and lacrimation are produced. Sneezing is violent and persistent; the nasal secretion is greatly increased and quantities of ropy saliva flow from the mouth. Nausea and vomiting may also occur. Mental depression may be so marked that the individual will have to be forcibly restrained to prevent self injury.

c. Diagnosis.—This is made from the history of exposure, and the relatively rapid spontaneous improvement which occurs despite the miserable appearance and condition of the individual.

d. Treatment.—Remove to pure air if possible. If this cannot be done, the mask must be worn between spells of actual vomiting and in spite of nausea and salivation. Inhalation of dilute chlorine from a small amount of bleach in a wide-mouth bottle is the most effective therapeutic measure. Inhalation of chloroform, ether, or alcohol vapor is less effective. Acetylsalicylic acid (aspirin) may be given to relieve the headache and general discomfort.

e. Prognosis.—No persistent lesion follows exposure to these agents. Ordinarily all symptoms disappear in about an hour.

15. Diphenylchlorarsine (DA).—The pathology, symptoms, diagnosis, treatment, and prognosis are similar to those of diphenylaminechlorarsine (DM).

Section VI

SCREENING SMOKES

Paragraph
General 16
White phosphorus (WP) 17
Titanium tetrachloride (FM) 18
Sulfur trioxide-chlorsulfonic acid solution (FS) 19
HC mixture (HC) 20

16. General.—The more important of these agents are white phosphorus (WP), sulfur trioxide-chlorsulfonic acid (FS), titanium tetrachloride (FM), and HC mixture (HC). In general these smokes

are not toxic in the ordinary field concentrations, but may be dangerous in the heavy concentrations formed at the immediate site of dispersion. The gas mask gives adequate protection against all of the screening smokes. FS smoke is more irritant and corrosive to the eyes, skin, and respiratory tract than is FM or HC.

17. White phosphorus (WP).—*a. Pathology.*—Particles of white phosphorus produce deep burns of the skin and perhaps underlying tissues, but systemic poisoning does not follow such injuries. The smoke is nontoxic, but long exposure to the fumes of white phosphorus may cause atrophy of the liver, osteitis, and bone necrosis.

b. Symptoms.—Skin burns are produced, the phosphorus particles continuing to burn in this tissue until air is excluded by a film of moisture or other material. In acute systemic poisoning from phosphorous vapor, jaundice may be produced, associated with an enlarged liver and gastro-intestinal disturbances. In chronic cases, cachexia and bone necrosis occur.

c. Diagnosis.—Diagnosis is made from the history of exposure, odor, and burns. Smoking or flaming particles may be present on or in the skin.

d. Treatment.—Smother the burn immediately with water, mud, or other air-impervious materials. Keep the burn under water until 5 to 10 percent copper sulfate solution can be applied which by chemical action forms a metallic coating on the particles, excluding air. The particles should be removed with forceps or hemostat under water, or in the air if the copper sulfate solution has been applied. In addition, all burned surfaces should be kept covered with wet dressings until it is certain that all particles of the agent have been removed. Further treatment is that of ordinary heat burns.

e. Prognosis.—Systemic poisoning is very rare under field conditions. The prognosis is the same as that of any heat burn of like degree and area.

18. Titanium tetrachloride (FM).—*a. Pathology.*—The liquid produces acid-like burns of the skin and eyes.

b. Symptoms.—The smoke generated by the liquid FM is unpleasant to breathe and may cause some irritation of the nose and throat, but it is not dangerous. However, exposure of the eyes to spray will cause conjunctivitis with lacrimation and photophobia. Acid skin burns are produced by contact with the liquid.

c. Treatment.—The eyes or skin should be washed freely with water and then with 2 percent sodium bicarbonate solution.

d. Prognosis.—No permanent injuries are produced.

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19. Sulfur trioxide-chlorsulfonic acid solution (FS).—*a. Pathology.*—Acid burns of skin or mucous membranes are produced by contact with the liquid.

b. Symptoms.—These are usually limited to an irritation of the skin, manifested by a biting and prickling sensation; direct contact with the liquid or concentrated vapor may cause corrosive burns of the skin and eyes.

c. Treatment.—The treatment of eye burns is most important. Irrigation with water should be instituted at once and continued for ½ hour, followed by repeated flushing with 2-percent sodium bicarbonate solution for 3 to 4 hours. Upon completion of this treatment a small amount of 2 percent aqueous mercurochrome solution should be dropped in the eye to detect possible corneal ulceration. If painful, 2 percent butyn may be instilled. Cod liver oil ointment or castor oil or yellow oxide of mercury ointment should then be instilled and the injured area covered with a light pad. Skin burns should first be washed with water and then with sodium bicarbonate solution; later treatment should be that of ordinary burns.

d. Prognosis.—The prognosis is good except for severe eye burns where the outlook depends on the degree of corneal ulceration.

20. HC mixture (HC).—*a. Pathology.*—Under field conditions there is insufficient absorption of the zinc salts or other components to produce systemic effects.

b. Symptoms.—There are no symptoms other than a slight suffocating sensation from high concentrations of the smoke.

c. Treatment.—None is needed.

d. Prognosis.—There are no lasting effects.

Section VII

INCENDIARY AGENTS

	Paragraph
General	21
Thermit (TH)	22
Combustible oil incendiaries	23
Combustible metal incendiaries	24

21. General.—*a.* The principal agents of this group are thermit (TH), white phosphorus (WP), combustible oil, and combustible metal incendiaries. While used primarily to cause fires in matériel, they will produce severe burns if they come in contact with the body.

b. Chemical fire extinguishers using carbon tetrachloride (pyrene) are apt to produce toxic gases, mainly phosgene, when this liquid comes in contact with flame or highly heated metal. In combating

incendiaries in inclosed spaces the danger from these byproducts must be kept in mind and the gas mask used if necessary.

c. The treatment of injuries from white phosphorus, which is also a screening smoke, is discussed in paragraph 17.

22. Thermit (TH).—*a. Pathology.*—Very deep burns of the skin and underlying tissues are produced by thermit.

b. Symptoms.—The symptoms are those of deep, exquisitely tender burns.

c. Treatment.—Particles of hot metal remaining on or in the skin must be cooled by flooding with as large a quantity of water as possible. Further treatment is that used for any other burn of like degree. The tannic acid treatment employed for mustard burns is recommended.

d. Prognosis.—This is the same as that for any heat burn of like degree and extent.

23. Combustible oil incendiaries.—*a. Pathology.*—Deep burns of the skin are produced, and inhalation of the combustion gases may cause pulmonary irritation.

b. Symptoms.—If actual contact with the skin occurs, deep burns with extensive coagulation of tissue may result. A productive cough may follow inhalation of the combustion fumes.

c. Treatment.—After the skin has been cleansed, burns from these oils should be treated in the same manner as ordinary burns.

d. Prognosis.—Serious with extensive burns and with pulmonary irritation.

24. Combustible metal incendiaries.—*a. Pathology.*—Deep burns are caused by contact with the hot fragments.

b. Symptoms, treatment, and prognosis.—These are the same as for other heat burns.

Section VIII

SYSTEMIC POISONS

	Paragraph
General	25
Hydrocyanic (prussic) acid	26
Arsine	27
Hydrogen sulfide	28

25. General.—In contrast to the lung irritants, vesicants, lacrimators, irritant smokes, and incendiaries, whose action on the body is predominantly local, there are certain other casualty-producing agents which produce their main injurious effects only after absorption into the body, and hence may be conveniently designated "systemic poisons." The most important of these agents are hydrocyanic acid,

arsine, and hydrogen sulfide. Adequate protection against these gases is afforded by the service mask.

26. Hydrocyanic (prussic) acid.—*a. Physical properties.*—Hydrocyanic acid is a clear, colorless, highly volatile liquid, and its vapor disperses rapidly in air. It is very soluble in water and alcohol and has an odor of bitter almonds or peach kernels. Aqueous solutions do not turn litmus red.

b. Pathology.—Hydrocyanic acid produces asphyxia by hindering the oxidative processes of the tissues, and depresses and paralyzes the central nervous system, beginning with the medulla.

c. Symptoms.—Following inhalation of high concentrations, vertigo, headache, palpitation, and dyspnoea come on very rapidly, followed shortly by coma, convulsions, and death. Low concentrations may produce headache, vertigo, and nausea.

d. Diagnosis.—The diagnosis is made from the odor of bitter almonds and the rapid onset of symptoms.

e. Treatment.—Remove the patient to pure air and give artificial respiration if necessary. Amyl nitrite fumes should be inhaled for 15 to 30 seconds every 3 minutes until sodium nitrite and sodium thiosulfate can be given intravenously. Sodium nitrite should be injected as a 1 percent solution slowly in 10 c. c. doses to a total of 50 c. c. an hour, and if necessary epinephrine should be employed against excessive fall of blood pressure. Between the nitrite injections, 20 c. c. doses of 5 percent sodium thiosulfate should be given intravenously and continued if necessary to a total of 500 c. c. In place of sodium nitrite, methylene blue may be given intravenously as a 1 percent solution in 1.8 percent sodium sulfate in 50 c. c. doses to a total of 200 c. c. If the patient becomes too greatly cyanosed from the methemoglobin produced by this treatment, blood transfusions should be given.

f. Prognosis.—The mortality of acute cyanide poisoning is very high, i. e., about 95 percent. Efficacy of treatment is limited by the rapid course of the poisoning and by the gravity of tissue asphyxia. If the patient survives an hour he will generally recover. After effects are very rare.

27. Arsine.—*a. Physical properties.*—This is a colorless, inflammable gas. It can be produced by the action of water on the arsenides of calcium, magnesium, and sodium. It has a characteristic garlic-like odor and metallic taste; however, the possibility exists that its inhalation in concentrations not detectable or recognizable by odor may cause toxic symptoms.

b. Pathology.—Arsine produces no irritation of the skin or mucous membranes. It is rapidly absorbed from the respiratory tract; the red blood cells are hemolyzed and part of the liberated hemoglobin is converted to methemoglobin. Destruction of red blood cells may be extreme. The kidneys may be blocked to various degrees by the debris of the red blood cells and by hemoglobin precipitation in the tubules. Anuria and uremia may result. Hepatitis is a common complication.

c. Symptoms.—In mild cases there may be lassitude, headache, and malaise. With increased exposure chills, nausea, and vomiting occur. In severe cases, anemia is the earliest prominent objective symptom, the red cell count frequently falling below 2 million. The methemoglobin formed produces a slatey cyanosis, this color changing later to that of jaundice, which is of hemolytic origin. The urine has a deep brown to red color. In fatal cases death is due to anemia or uremia.

d. Diagnosis.—The diagnosis is made from the history of exposure and the development of anemia, hemoglobinemia, and hemoglobinuria without other apparent cause, such as blackwater fever and paroxysmal hemoglobinuria. Both of the latter conditions occur only sporadically, and not in a number of simultaneous cases as would probably be the case following the projection of arsine against personnel. The possible causative role of blackwater fever should be investigated from the viewpoints of history of residence, malaria, recent quinine medication, high fever and blood examinations, while the etiological significance of paroxysmal hemoglobinuria is based on the previous medical history (syphilis) and exposure to cold or abnormal physical exercise.

e. Treatment.—(1) The immediate treatment consists of removal to pure air or masking. Ferric hydrate paste (see par. 5*d*, app.) diluted with water or magma ferri hydroxidi (USP) may be given orally, though with even less assurance of real benefit than after other types of arsenic poisoning. Either antidote should be followed by an effective cathartic. Further treatment is directed mainly against the anemia and the toxic action on the kidney. If the red cell count falls below 4,000,000, or the hemoglobin below 60 percent, 500 c. c. of blood should be given by transfusion; while hemolysis of new cells is probably slight a few hours after exposure, this destructive process may continue for several days, making repeated transfusions necessary. The loss of new cells is not accompanied by loss of fluid as in hemorrhage, and care should be taken to avoid the injection of excessive amounts of fluid.

(2) Diuretic fluids should be given by mouth freely up to about 4,000 c. c. in 24 hours. Administration should be by rectum if fluids are not retained by mouth. Organic mercurial diuretics should not be given.

(3) Alkalies will aid diuresis and may prevent precipitation of hemoglobin in the kidney tubules. This precipitation of hemoglobin is at least one, if not the most important, factor in the kidney blockage. Sufficient alkali should be given to maintain an alkaline urine. Divided doses of sodium bicarbonate up to 20 grams daily, or, preferably, sodium citrate or acetate up to 50 grams daily are suggested. Administration of alkalies should be started as early as possible and continued in amounts required to maintain an alkaline urine as long as excretion of hemoglobin lasts.

(4) Glucose is indicated especially if liver involvement occurs. It may be given with or without insulin, and intravenously if necessary.

(5) During convalescence the anemia should be combated by the use of iron and liver extract. Resumption of physical activity should be gradual. The diet should be light as long as evidence of liver or kidney damage persists.

f. Prognosis.—The mortality is about 30 percent. Recovery may occur even with severe cases; others may die within 2 to 6 days. Marked anemia and anuria are grave signs.

28. Hydrogen sulfide.—*a. Physical properties.*—This is a colorless gas which is heavier than air. It has an odor of rotten eggs and may be detectable at a harmless concentration, but the sense of smell becomes dulled quite rapidly and even high concentrations may not be recognized.

b. Pathology.—Hydrogen sulfide produces local irritation of eyes, nose, and throat, and high concentrations may produce pulmonary edema and paralysis of the respiratory center.

c. Symptoms.—These consist of local irritation of the eyes, exposed mucous membranes, and respiratory tract. Pulmonary edema may result from the respiratory irritation. Effective concentrations produce an increased, panting respiration, quickly followed by unconsciousness and cessation of breathing which may be accompanied by convulsions.

d. Diagnosis.—This may be made from the odor, rapid loss of consciousness, cessation of respiration before the heart fails, and the blackening of lead acetate paper or of a moistened silver coin by exposure to the expired air.

e. Treatment.—Remove to fresh air and apply artificial respiration for long periods even though the procedure may for some time appear

hopeless. Inhalation of oxygen-carbon dioxide mixtures should also be employed.

f. Prognosis.—In high concentrations, hydrogen sulfide approaches hydrocyanic acid in toxicity.

SECTION IX

INCIDENTAL GASES

Paragraph
General 29
Carbon monoxide 30
Nitric (nitrous) fumes 31
Ammonia 32

29. General.—*a.* This group includes certain gaseous compounds, such as carbon monoxide, nitric fumes, and ammonia, which may be met with indirectly in military operations as the result of fires or shell explosions in confined or poorly ventilated spaces, or in the bombing of industrial installations. Carbon monoxide, and to a lesser extent nitric fumes, are combustion products which may remain for some time in freshly formed shell holes, and may seep into underground shelters and passages. Carbon monoxide may also be formed in collective shelters from incomplete combustion in heating devices such as charcoal braziers. Ammonia may be liberated in refrigeration plants accidentally or as the result of enemy bombing activities.

b. Protection against incidental gases.—The service mask canister furnishes protection against nitric fumes, but not against carbon monoxide or ammonia, for which special canisters are necessary. This is also true of collective protection devices.

c. Oxygen deficiency.—(1) Ordinary air normally contains about 21 percent oxygen. However, the proportion of oxygen may be reduced to a dangerous degree in closed or poorly ventilated spaces such as shelters, compartments, or underground tunnels, by human consumption, combustion by fire, or dilution with other gases such as carbon dioxide, methane, etc. The reduction in the partial pressure of oxygen in rarefied air at high altitudes, while mainly of concern to aviation, may also be a factor deserving consideration in land operations at high altitudes. When the oxygen content of air falls below 17.5 percent a lighted candle will be extinguished, and this is a good test for the safety of the air as far as the oxygen content is concerned, since human symptoms of oxygen deficiency are not noticeable until an even lower oxygen level has been reached.

(2) It is obvious that the service gas mask is of no value in oxygen deficiency, and that in this condition either an oxygen breathing

apparatus or a mask with a hose reaching the outside air is necessary. If the hose is longer than 25 feet a pump will be needed to secure an adequate supply of air, but otherwise a satisfactory air current will be maintained through the respiratory movements and the operation of the inlet and outlet valves as in the service mask.

30. Carbon monoxide.—*a. Physical properties.*—Carbon monoxide is a colorless, odorless gas, much lighter than air, into which, however, it diffuses rapidly.

b. Occurrence in military operations.—Carbon monoxide is formed by gun blasts, bursting shells, and internal combustion engine exhausts. Dangerous concentrations are apt to occur in confined spaces such as poorly ventilated gun turrets or emplacements, in mining operations, or in recently formed shell holes.

c. Pathology.—Asphyxiation is produced by the union of carbon monoxide with hemoglobin, for which it has an affinity 300 times greater than oxygen, thus replacing the latter. (Carbon monoxide can in turn be displaced from the compound by a sufficient concentration of oxygen.) As a result of oxygen deficiency, nervous system changes occur. Post mortem examinations reveal little beyond the characteristic cherry-red color of the blood and mild hemorrhagic changes in the brain.

d. Symptoms.—The symptoms progress from headache, throbbing of the temples, vertigo, yawning, dyspnoea, dullness, and lack of visual acuity to the development of bright pink patches on the skin, marked weakness and coma with subnormal temperature, and low tension pulse and death.

e. Diagnosis.—The diagnosis is made from the cherry-red color of the blood (compared with the color of normal blood) and bright red patches on the skin.

f. Protection.—The service canister does not protect against carbon monoxide, a special canister being required. Adequate ventilation should be provided for all inclosed spaces where carbon monoxide may be produced. The safety of the air in a space may be tested by introducing a cage containing a small animal such as a mouse, rat, or much better, a canary. The air will be safe for man to breathe for at least as long as these animals remain unaffected in such air.

g. Treatment.—Remove to pure air, give pure oxygen or oxygen carbon dioxide mixture, and artificial respiration if necessary. Rest, blankets, and warm drinks are also indicated. Blood transfusions are valuable in desperate cases.

h. Prognosis.—The longer the period of coma the less the chances of recovery. Most of the cases recover with early treatment. Tachy-

cardia and dyspnoea may continue for months along with central nervous disturbances ranging from simple neuritis to mental deterioration.

31. Nitric (nitrous) fumes.—*a. Physical properties.*—Nitric fumes or vapors consist mainly of nitrogen dioxide (NO) and nitrogen tetroxide or peroxide (NO_2 and N_2O_4). They are orange-yellow or reddish-brown in color, very soluble in water, and react with water and oxygen to form nitrous and nitric acids.

b. Occurrence in military operations.—These gases are produced wherever nitro explosives are used and incompletely detonated or subjected to slow combustion in confined quarters. Such conditions may occur in gun pits, armored cars, tanks, ship magazines, and in mining and tunneling operations. Nitric fumes may also be produced by the action of dilute nitric acid on copper.

c. Pathology.—Irritation of the lungs is produced, followed by the development of pulmonary edema; some local ulceration of the lungs may be caused by nitric acid, formed where the fumes come in contact with moist areas in these organs. In general the pathological findings are similar to those produced by phosgene.

d. Symptoms.—There may be a slight conjunctivitis, rhinitis, and pharyngitis, followed by a latent period of 4 to 8 hours with very few symptoms or signs; it is the absence of marked sensory irritation and the latent period which are responsible for exposed individuals failing to realize the serious danger following inhalation of these fumes. There may be yellowish discoloration of the mucous membranes only if the gaseous concentration is very great. The picture of lung irritation and edema, similar to that following phosgene, is last to develop.

e. Diagnosis.—The diagnosis is made from the history of exposure, pungent odor, yellowish discoloration of the exposed mucous membranes, irritation of nose, throat and lungs, and later pulmonary edema.

f. Treatment.—Same as for phosgene.

g. Prognosis.—Casualties usually die within 24 to 48 hours from pulmonary edema. A fatal broncho-pneumonia may complicate other cases; convalescence is prolonged especially when complicated with simultaneous exposure to carbon monoxide.

32. Ammonia.—*a. Physical properties.*—Ammonia is a colorless gas with a very pungent, characteristic odor. It is only about half as heavy as air and is exceedingly soluble in water.

b. Occurrence in military operations.—This gas is not used in warfare but may be encountered under the circumstances mentioned in paragraph 29.

c. Pathology.—Exposure to high concentrations of ammonia produces a prompt and violent irritation of the eyes and respiratory tract. There may be edema of the glottis and a yellowish, purulent, pseudomembranous laryngitis.

d. Symptoms.—The symptoms following inhalation of high concentrations consist of a violent burning pain of the eyes and nose accompanied by lacrimation and sneezing, pain in the chest, severe coughing, spasm of the glottis, and signs of a terminal lung infection. Often there is temporary reflex stoppage of respiration, and asphyxia may result from the spasm and edema of the glottis or from swelling of the mucous membrane of the larynx and trachea. Concentrations of 0.1 percent are insupportable for man. Rubefaction and sometimes vesication of the skin may follow direct contact with liquid ammonia or ammonium hydroxide.

e. Treatment.—Following inhalation, first-aid treatment consists of prompt removal to pure air, and artificial respiration if necessary. Inhalation of the fumes of weak acetic acid or vinegar may be of some benefit. Later measures are directed toward the prevention or treatment of bronchitis and pneumonia.

f. Prognosis.—The mortality is high following the inhalation of high concentrations; with lower concentrations recovery is usually rapid, although a somewhat persistent bronchitis may result.

SECTION X

MEDICAL SERVICE IN CHEMICAL SITUATIONS

Paragraph
General 33
Provisions for chemical casualties 34

33. General.—Medical Department installations are subject to exposure to chemical agents in the same manner as are other military establishments occupying similarly located areas, i. e., to mixed chemical agents from artillery fire, and vesicant and incendiary agents from airplane projection. The necessary precautions for providing individual and collective protection should therefore be taken well in advance of possible exposure to such attacks.

34. Provisions for chemical casualties.—*a.* Contact with patients whose bodies or clothing have been contaminated with liquid vesicants will result in vesicant injury to all unprotected attendants so exposed. Litter bearers and aid station personnel must wear masks and protective aprons or other protective clothing when tending such casualties.

b. When casualties from persistent vesicant agents arrive at medical

installations, special precautions should be taken for their reception and disposal in order to guard against exposure of medical personnel, patients, and equipment. Well in advance of the reception of such casualties, arrangements should be made for their isolation, decontamination, treatment, or other disposal (see chart, par. 4, app.). Such measures include special wards, separate medical personnel, and individual protective equipment. The necessity for decontamination of litters and ambulances transporting such casualties should always be borne in mind, and, if possible, provision should be made for oxygen administration in ambulances.

c. As pointed out earlier in this manual, in the case of certain chemical agents a latent period may intervene between the time of exposure and the first appearance of symptoms. During this latent period, rest is of primary importance. The question may arise as to whether exposed troops are to be given the status of casualties during this latent period, and when such casualty status is to be initiated. In combat the decision will rest with the responsible commanding officer whether to require such troops to continue on duty at the risk of increasing the danger of subsequent pathological manifestations, even to the point of death, or to relieve them from duty in order that they may come under appropriate medical care as early as practicable. It is conceivable that only in desperate and decisive situations will an organization commander feel justified in subjecting his men to the increased hazard of remaining in combat during this latent period. It would appear to be the duty of the responsible medical officer on such occasions thoroughly to inform the organization commander of the additional hazards involved in order that he may make his decisions with all of the facts in mind. Similar action with regard to animal casualties is indicated.

Section XI

SPECIAL CASES

	Paragraph
General	35
Protection for head-injury casualties	36
Vesicant-contaminated wounds	37
Employment of anesthesia for chemical casualties	38

35. General.—In the care of chemical casualties certain conditions may arise which will indicate the use of special supplies, equipment, or methods of treatment. These will not always be available, and in such conditions medical officers will exercise their ingenuity in the application of such measures as are best adapted to the special situation. Examples include cases where wounds of the head and face

preclude the wearing of the service mask, casualties where wounds are contaminated with vesicants, and the application of general anesthesia in gas casualties.

36. Protection for head-injury casualties.—A special head mask may be required for patients in gas-contaminated localities where injuries, dressings, or personal discomfort may interfere with the utilization or proper functioning of the service mask. This mask will be in the form of a large hood sufficient to cover the entire head and shoulders and compensate for any type of extensive head dressing. The purified air supply will be furnished ordinarily by hand bellows through an attached canister or by a collective protector; if neither of the latter is available, the tube and canister of the service mask should be connected to the inlet tube of the special mask, and the bottom of the hood must be fastened securely to prevent inward leaking. Since there is more dead space in the special head mask than in the service mask, air must be furnished by the bellows or other means as soon as possible.

37. Vesicant-contaminated wounds.—*a.* It is presumed that by the time gas casualties have reached surgical operating installations their skin and clothes will have been decontaminated. Operating personnel, however, should always consider the possibility that chemical contamination exists in deep, penetrating, or extensive wounds, such as those of the abdomen, where exploration may have to be done. If there is a strong odor of the vesicant about the wound, all members of the team will have to wear masks and use some form of protective clothing, especially if the operating room is in small, confined, or poorly ventilated quarters. Instruments should be decontaminated by boiling in several changes of sodium carbonate solution or by the use of CWS noncorrosive decontamination agent, chloramine-T, dichloramine-T, or Dakin's solution. Only clean, uncontaminated instruments and fresh gloves should be used after the contaminated tissue has been debrided. Since vesicants may penetrate the surgical rubber glove, it is suggested that the hands be covered with protective ointment before the gloves are put on. Special protective gloves are very large and awkward for operating work but may be worn if the circumstances warrant.

b. If the vesicant has penetrated deeply into the wound, the latter must first be irrigated repeatedly with 0.5 percent sodium hypochlorite (Dakin's solution), followed by debridement. In subcutaneous and muscle tissues contaminated by arsenicals, extensive debridement must be performed to prevent or minimize arsenic intoxication. Where wounds of the abdominal cavity are contaminated by arseni-

cals, systemic poisoning will follow, and general therapy in the form of administration of intravenous fluids, especially glucose solutions, must be started at once. As far as is possible, the surgeon and his assistants should avoid touching heavily contaminated areas directly with their gloves, using instruments instead for this purpose. Special precautions must be taken for disposal of the discarded contaminated tissues. Nonvesicant casualties should not be touched until the surgeon and assistants have decontaminated themselves.

38. Employment of anesthesia for chemical casualties.—*a.* When anesthesia is necessary for wounded casualties who have also been exposed to lung irritant gases, local infiltration or nerve block are the methods preferred, but intravenous, spinal, or rectal routes may be employed. At times these methods may be neither available nor desirable, and in some cases within certain restrictions it may be practicable or necessary to use inhalation anesthesia.

b. During the latent phase of pulmonary irritation, surgical interference must be confined to life-saving measures such as those necessary in laryngeal-tracheal injuries, hemorrhage, etc., and if anesthesia is necessary its use should be as brief as possible; when inhalation anesthesia is definitely indicated, ether should be avoided in favor of less irritant anesthetics. In situations where the use of spinal anesthesia may appear logical, it should be remembered that it may predispose to the development of cardio-vascular collapse which so often occurs following exposure to pulmonary irritants.

c. If pulmonary edema has developed, the treatment of this condition will be more important than surgical treatment of any accompanying injuries, and general anesthesia is absolutely contraindicated. If the patient survives the edema, any surgical interference should be deferred for at least 48 hours.

d. When it is impossible to furnish collective protection for surgical operating installations in areas liable to chemical attack, it is best to defer setting up such installations until the military situation definitely requires such action. When exposure is imminent the patients and the entire operating personnel must be protected by masking. While the surgeon and his assistants can operate when masked, it is not ordinarily possible similarly to protect a patient requiring inhalation anesthesia. When operations are necessary despite the presence in the air of chemical agents, and general anesthesia is mandatory, a closed system should be used, if available, to furnish both oxygen and the anesthetic.

SECTION XII

CONTAMINATED FOOD AND FORAGE

Paragraph
General 39
Nature of chemical decontamination 40
Reclamation of contaminated supplies 41

39. General.—Consumption of chemically contaminated food, forage, grain, and water will produce irritation of the gastro-intestinal tract and other systemic toxicological effects. While such unprotected supplies under suspicion of contamination by chemical agents are normally discarded, in case there is a shortage of food special instructions from a medical officer will govern as to its decontamination (par. 26, FM 21–40). In accordance with this responsibility medical officers should, in general, be guided by the procedures outlined below. Special procedures relating to the purification of water will be available.

40. Nature of chemical decontamination.—Of the many chemical agents used in warfare, those of most concern in the contamination of food stuffs will include the vesicants and arsenicals. These and certain other agents are highly soluble in fats, and will thus be readily absorbed by foods of high fat content, and diffusing throughout the material will be difficult or impossible to remove. Coagulation of protein by agents which are acid or form acids in foods of high protein content may limit diffusion of the agent into the interior. Hydrolysis of certain acid and acid forming gases in foods of high water content will allow diffusion of decomposition products throughout the mass and greatly impair the taste. Foods of low water and fat content will be relatively less receptive to contamination by chemical agents and less difficult to decontaminate.

41. Reclamation of contaminated supplies.—*a.* The most effective and practical measures for purifying food, forage, and grain when contaminated with chemical agents include trimming of exposed surfaces, complete aeration or ventilation, and hydrolytic procedures, such as boiling in water or washing in dilute (5 percent) bicarbonate of soda solution. Even these measures may be ineffective if the decomposition products are themselves toxic, as in the case of lewisite.

b. In general, food, forage, or grain exposed to low vapor concentrations of chemical agents can be reclaimed by the procedures listed above. It is impracticable to reclaim provisions that have been heavily contaminated by vapors, droplets, or dusts, especially of the vesicants, arsenicals, or those agents which upon hydrolysis split off other toxic compounds. Highly contaminated supplies, such as those

on which chemical agents can be seen with the unaided eye, should be considered spoiled and their decontamination or purification impracticable.

c. While recognizing difficulties of decontamination procedures, it is nevertheless realized that it may be necessary or even mandatory to use these measures under certain tactical or economic conditions, particularly when there is a marked shortage of foodstuffs or where great losses of salvable products are involved. After reclaiming foods, especially those of high fat content, contaminated with the highly acid gases, the taste of such products may be greatly impaired, although the energy content of the food is not altered. It must be emphasized that the use of any decontamination procedure will be greatly complicated when foodstuffs have been exposed simultaneously to the action of more than one type of chemical agent.

d. When it is necessary to use animals for food following their exposure to lung irritant gases, the carcass meat may be consumed if slaughtering has been done in an approved manner, even while pulmonary injury is still active, provided the other internal organs are normal, since even animals fatally poisoned by these gases die before the agent spreads throughout the carcass meat. Portions of carcass meat directly under the skin areas affected by vesicants should be discarded.

APPENDIX

1. Tabulated data on certain chemical agents.

LUNG IRRITANTS

Name	PHOSGENE (Carbonyl chloride; "palite")	CHLORPICRIN (Trichlornitromethane; "vomiting gas")	CHLORINE	NITRIC VAPORS (NO, NO_2, N_2O_4)	AMMONIA
CWS symbol	CG	PS	Cl	None	None
Method of recognition.	Colorless gas, odor of cut corn, ensilage, or musty hay; renders smoking unpalatable.	Colorless, volatile liquid; vapor has odor of fly paper or licorice.	Greenish-yellow gas; odor like that of bleach; makes smoking unpalatable.	Reddish-brown fumes, with pungent, aromatic odor.	Colorless gas, with sharp penetrating odor.
Tactical uses	Casualty agent, delivered from cylinders, projectors, mortar, and artillery shell.	Harassing and casualty agent, in grenades, mortar and artillery shell, airplane spray, and bombs.	Casualty agent, dispersed from cylinders and projectors.	No tactical or military use. *Incidental* to combustion of nitro-compounds and explosives.	No tactical or military use. *Incidental* to manufacturing or refrigerating accidents.
Symptoms and effects.	Coughing and lacrimation, pain and sense of constriction of the chest, dyspnoea, later pulmonary edema.	Similar to phosgene, but more irritant and lacrimating. More apt to produce vomiting. Less toxic than	Similar to phosgene, but more irritant and less toxic. Coughing more pronounced than with phosgene or	Similar to phosgene. Irritation of nose, throat, and lungs, later pulmonary edema.	Prompt, marked irritation of eyes, nose, throat, and lungs; effective concentrations may cause temporary reflex

		phosgene. Pulmonary edema may follow.	chlorpicrin. Pulmonary edema may follow.		stoppage of respiration. Bronchitis, laryngitis, and pneumonia may follow.
First aid treatment.	Absolute rest and warmth, hot drinks; venesection in blue stage; oxygen administration. Evacuate in prone position.	Same as for phosgene; in addition wash splashes on skin with sodium carbonate solution or alcoholic sodium sulfite solution.	Same as for phosgene.	Same as for phosgene.	Artificial respiration, inhalation of weak acid (acetic) vapor.
Protective measures.	Service gas mask; collective protection shelters.				*Service gas mask canister does not protect against ammonia*; special canister necessary.

VESICANTS

Name	MUSTARD (Bisbetachlorethysulfide; "Yperite" "Senfgas")	LEWISITE (Betachlorvinyldichlorarsine)	ETHYLDICHLORARSINE ("Dick")
C.W.S. symbol	HS	M-1	ED
Method of recognition.	Dark brown to straw-yellow oily liquid, odor of vapor like that of garlic, horseradish, mustard, or onions.	Colorless to brown liquid; odor of vapor like that of geraniums; immediate sneezing and nasal irritation.	Pungent odor; rapid, intense irritation of eyes and nose; sneezing.
Tactical uses	Casualty agent delivered from mortar, howitzer and artillery shell, airplane spray, or bombs.	Casualty agent, projected in same manner as mustard.	Casualty and harassing agent, from artillery and mortar shell, and airplane spray.
Symptoms and effects.	*Vapor:* Delayed irritation and inflammation of eyes and conjunctiva; delayed erythema of skin, with or without blistering; bronchitis; pneumonia. *Liquid:* Prompt irritation, inflammation of eyes; corneal lesions may result in blindness; inflammation and erythema of skin with severe vesication.	*Vapor* and *liquid:* Prompt irritation and inflammation, erythema, marked vesication of the skin; severe eye lesions perhaps followed by blindness; bronchitis, and pneumonia from inhalation of vapor; systematic arsenic poisoning may occur from large burns.	Irritation, erythema, and vesication of skin; inflammation of eyes and conjunctiva; irritation of respiratory tract with marked sneezing.
First-aid treatment	*Skin:* If liquid, remove excess with cloth sponge. Wash with alcohol, kerosene, or carbon tetrachloride, or hot water and soap. Apply bleach paste or protective ointment and remove by wiping or	*Skin:* If liquid, remove excess with cloth sponge. Wash with soap and water, then with sodium hydroxide solutions *followed by water.* If large skin areas are involved, the skin should be excised. Multiple	General treatment same as for lewisite; excision or cupping not necessary unless lesions are extensive and severe.

	washing. Treat burns as ordinary heat burns. *Eyes:* Irrigate with 2 percent sodium bicarbonate or saturated boric acid. Instill 2 percent butyn sulfate for pain. Do not cover or bandage.	incision with cupping may be a valuable procedure. *Eyes:* Same treatment as for mustard injuries.
Protective measures	Service gas mask; protective clothing; protective ointment; collective protection.	

	LACRIMATORS			STERNUTATORS (Toxic smokes)	
Name	TEAR GAS SOLUTION (Chloracetophenone, chlorpicrin, chloroform)	CHLORACETOPHENONE	BROMBENZYLCYANIDE	DIPHENYLAMINECHOLRARSINE (Adamsite)	DIPHENYCHLORARSINE
CWS symbol	CNS	CN	CA	DM	DA
Method of recognition.	Straw-colored solution, with sweetish, flypaper odor.	Colorless crystals, with odor of apple or locust blossoms.	Yellowish-brown crystals, or in brown solution; odor pungent and like that of sour fruit.	No odor: recognizable only by onset of symptoms. Produces canary-yellow smoke haze.	
Tactical uses	Harassing agent used in mortar shells, grenades, air bombs, and airplane sprays.	Harassing agent used in candles, grenades, mortar and artillery shell, airplane sprays, and bombs.	Harassing agent; can be used in artillery shell and airplane spray.	Harassing agent used in candles, destroyer smoke stack, burning type munitions.	Harassing agent used in burning type munitions.
Symptoms and effects.	Immediate lacrimation, photophobia, prickling or itching of the skin.	Similar to CNS, with perhaps less action on the skin.	Severe lacrimation and nasal irritation.	Sneezing, with burning, aching pains in nose, throat, chest, sinuses, followed by headache, sinus pains, nausea, and often mental depression.	
First-aid treatment.	Face wind with eyes open; in more severe cases, wash eyes with 2 percent sodium bicarbonate or saturated boric acid solution. *Do*			Inhale chlorine fumes through nose and mouth from bleach bottle; acetylsalicylic	

	not rub or bandage eyes. Persistent pain may be relieved by instilling 2 percent butyn sulfate. If necessary, skin may be washed with sodium bicarbonate solution.	acid may be taken orally to relieve pain. Physical restraint may be necessary to prevent self-injury.
Protective measures.	Service gas mask; collective protection.	Service gas mask; collective protection.

SYSTEMIC POISONS

Name	HYDROCYANIC ACID (HCN; prussic acid)	ARSINE ($As H_3$)	HYDROGEN SULFIDE (H_2S)	CARBON MONOXIDE (CO)
CWS symbol	None	None	None	None
Method of recognition.	Colorless, volatile liquid; odor of vapor like that of peach kernels, or oil of bitter almonds.	Colorless, inflammable gas, with nauseating, garlic-like odor.	Colorless gas, with odor of rotten eggs.	Colorless, odorless gas; recognizable only by onset of symptoms.
Tactical uses	Casualty agent. Used in artillery shell.	Casualty agent. Potential use in cylinders, shell and bombs, or in form of arsenical powder which would liberate arsine on contact with moisture.	No tactical use. Is *incidental* to chemical manufacturing, analyses, and putrefactive processes.	No tactical use. Is *incidental* to bursting of explosive shell, or various types of combustion, i. e., gun blasts, engine exhausts, etc.
Symptoms and effects.	Low concentration of vapor may cause giddiness or headache; effective vapor concentrations or the liquid rapidly produce convulsions, unconsciousness, or death from tissue asphyxia or medullary paralysis.	Shivering, weakness, giddiness, nausea, vomiting, headache, gray color, collapse; hemolysis, anemia, anuria, uremia.	Irritation of eyes, nose, respiratory tract; bronchitis; high concentrations cause unconsciousness and death.	Dizziness, headache, weakness, nausea and vomiting, feeling of constriction in thorax, followed by drowsiness, visual disturbances, stupor, unconsciousness, weakened pulse and respiration, and death.
First-aid treatment.	Inhale amyl nitrite. Give artificial respiration if necessary. Sodium ni-	Absolute rest; evacuate in prone position. Ferric hydroxide or arsenic	Absolute rest; artificial respiration. Give oxygen-carbon diox-	Artificial respiration, administration of oxygen or oxygen-carbon dioxide;

	trite, sodium thiosulfate, or methylene blue should be given intravenously.	antidote by mouth may be tried. Give large amounts of fluid, blood transfusions, and try to promote diuresis.	ide and blood transfusions.	venesection with transfusion of healthy blood may be valuable.
Protective measures.	Service gas masks; collective protection.			*Service gas mask canister does not protect against carbon monoxide;* special canister necessary.

INCENDIARIES

Name CWS symbol	WHITE PHOSPHORUS WP	THERMIT TH	ELECTRON BOMB [1] None
Method of recognition	Smoke has no odor. Element burns only in presence of oxygen (air), so action can be stopped with water, oil, etc. Will reignite on access of air. Solid has odor of matches.	On ignition forms red, molten mass of metal giving off extremely hot reddish-yellow flame, extinguishable with water.	On ignition burns with brilliant white, intensely hot flame, casing of shell being consumed. Water only increases combustion and may cause explosion in closed space.
Tactical uses	Screening and casualty agent. Used in grenades, mortar and artillery shell, airplane bombs.	Incendiary agent for matériel. Used in airplane bombs (and perhaps shell).	Incendiary agent for matériel. Used in airplane bombs.
Symptoms and effects	Severe burns depending on size of particles and length of contact.	Same as with white phosphorus.	More severe penetrating burns than with white phosphorus or thermite, as it continues to burn in tissues in presence of moisture.
First-aid treatment	Cover burning surfaces with water, or preferably 5 percent copper sulfate solution, and remove phosphorus particles with forceps. Further treatment same as for ordinary burns.	Spray burning areas with water, and remove pieces of material. Further treatment same as for ordinary burns.	Remove burning material. Treatment of injuries that of ordinary burns.
Protective measures	Fireproof clothing or shelters are the only protective measures.		

[1] The electron bomb is a magnesium case filled with fast-burning thermit. The thermit sets fire to the magnesium case which is the effective incendiary material in this bomb. Electron bombs are being widely used in the present European conflict.

2. Diagnosis.—*a. Questions to be asked patient to establish diagnosis.*

(1) When were you exposed?
(2) Where were you and what were you doing?
(3) Do you know what agent was used?
(4) What did the agent smell like?
(5) Was it used as a gas or spray?
(6) How long was it before you put on your mask?
(7) When did you remove your mask?
(8) What ill effects did you first feel?
(9) How do you feel now?
(10) What treatment have you had?
(11) Have you any other wounds?
(12) Have you been sick lately?
(13) Did you walk or ride here?
(14) How does a cigarette taste to you?

b. Differential diagnosis of lung irritant casualties.

Agent	PHOSGENE (CG)	CHLORPICRIN (PS)	CHLORINE (Cl)	MUSTARD (HS)	LEWISITE M-1	ETHYLDICHLORARSINE (ED)	NITRIC FUMES
Odor	Like cut corn, moldy hay	Like licorice, sweetish	Pungent	Garlic	Geranium	?	?
Appearance of casualty.	Normal, or cyanotic or gray.	Lacrimation, nausea, emesis.	Face flushed; very uncomfortable.	May be associated skin burns.	Same as for HS.	Sneezing, lacrimation, skin burns.	Normal or cyanotic.
Latent period	Long.	Moderate.	Short.	Long.	Short.	Short.	Long.
Cyanosis	Marked.	Not marked early.	Marked.	Not marked early.	Not marked early.	Not marked.	Marked.
Engorgement of neck veins.	Present except in gray form.	Not present early.	Present.	Not present at first unless there is pulmonary edema.	Same as for HS.	Not present early.	Present.
Sputa	Abundant, frothy, bloody.	Sparse at first unless exposure was severe.	Abundant, frothy, bloody.	Sparse unless pulmonary edema; later mucopurulent.	Same as for HS.	Sparce.	Abundant, bloody frothy.
Cough	Violent.	Moderate.	Violent paroxysmal.	Not marked early unless pulmonary edema.	Same as for HS.	Same as for HS.	Violent.

Chest pain	Not marked.	Not marked.	Severe.	Not marked early unless pneumonia.	Not marked early unless pneumonia.	Burning type.	Not marked.
Auscultation of chest.	Moisture throughout.	Moisture at first in bronchi, later throughout.	Moisture throughout.	Moisture chiefly in bronchi, later signs of consolidation.	Same as for HS.	Moisture chiefly in bronchi.	Moisture throughout.
Secondary broncho-pneumonia.	Uncommon.	Uncommon.	Uncommon.	Common.	Common.	Uncommon.	Uncommon.
Prognosis	Poor with blue or gray: better if survives 48 hours.	Good unless pulmonary edema.	Good unless pulmonary edema.	Good if no broncho-pneumonia and no extensive burns.	Same as for HS.	Rarely fatal under field conditions.	Good if no pulmonary edema.
Characteristic post-mortem findings.	Alveolar involvement, chiefly pulmonary edema.	Irritation, chiefly of upper bronchial tree; may be some alveolar damage.	Damage to upper bronchi and some alveoli; pulmonary edema.	Purulent broncho-pneumonia with ulcerations in upper bronchial tree. Skin burns.	Purulent broncho-pneumonia with ulcerations in upper bronchial tree. Skin burns. Arsenic in skin and viscera.	Toxic changes in viscera; arsenic present.	Pulmonary edema.

(1) Prognosis and treatment are governed to some extent by the type of causative agent as determined from the differential diagnosis, which, however, may be difficult or impossible to make after signs or symptoms of pulmonary edema have developed.

(2) Occasionally death may occur by reflex action immediately after the first inhalation of heavy concentrations of the lung irritants, particularly chlorine; on the other hand it should always be borne in mind that more often no immediate symptoms occur following inhalation of these agents.

c. Differential diagnosis of vesicant casualties.

Name	MUSTARD (HS) (Bisbetachlorethylsulfide)	LEWISITE (M-l) (Chlorvinyldichlorarsine)	ETHYLDICHLORARSINE (ED)
Odor	Garlic.	Geranium.	Biting.
Onset	Slow.	Rapid.	Rapid.
Pain at onset	Slight or none.	Severe.	Slight.
Erythema around blister.	Marked.	Slight.	Slight.
Early color of blister.	Clear.	Opaque.	Opaque.
Degree of blistering.	Severe.	Very severe.	Moderate.
Later secondary infection of blisters.	Common.	Uncommon.	Uncommon.
Duration of healing.	Long.	Moderately long.	Short.
Associated systemic poisoning.	Uncommon (toxemia).	Common (arsenical).	Fairly common. (arsenical).
Pigmentation after healing.	Common.	Uncommon.	Uncommon.
Prognosis	May be fatal if broncho-pneumonia occurs.	May be fatal from arsenical poisoning.	Rarely fatal under field conditions.
Persistency	Most persistent.	Persistent.	Less persistent.

3. Chemical memoranda for the medical officer.—*a. What to do.*—(1) Learn gas discipline yourself.

(2) Protect yourself from contamination.

(3) Give treatment as rapidly as possible.

(4) Evacuate your chemical casualties as rapidly as possible.

(5) Make every effort to know the exact etiologic agent or agents, since these may be mixed together.

(6) Examine the casualty for nongas wounds.

b. What not to do.—(1) Do not allow phosgene cases to be up and about.

(2) Do not give artificial respiration to acute phosgene casualties.

(3) Do not use cocaine in mustardized eyes.

(4) Do not forget that there may be latent periods with many of the chemical agents.

(5) Do not use contaminated gloves on a nonchemical casualty.

(6) Do not fail to look at the eyes of all casualties.

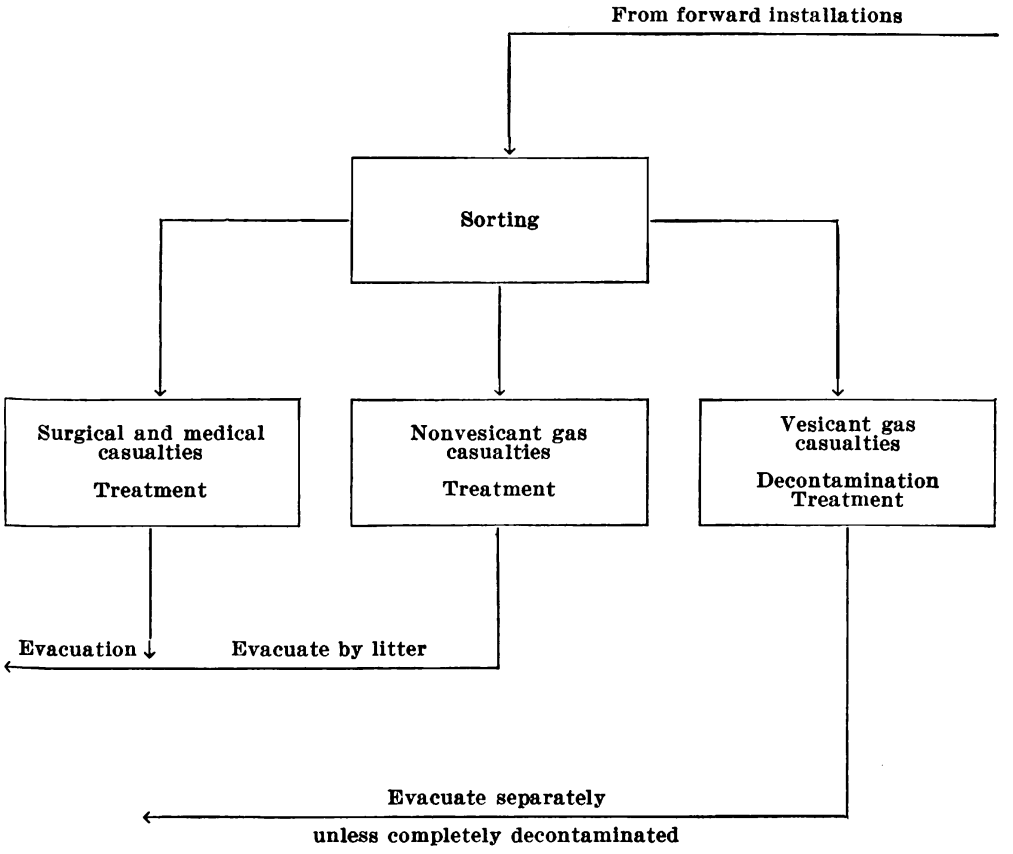
(7) Do not give morphine to phosgene cases.

c. First-aid cautions.—(1) The primary step in first aid procedure is to remove the casualty from the contaminated environment; if this is not possible, he should be properly masked or placed in a collective protection shelter.

(2) Clothing which has been splashed with a liquid agent should be removed immediately.

(3) Affected eyes should never be rubbed or bandaged but should be shielded from light.

4. Chart showing sorting and evacuation of casualties and necessary separation of vesicant casualties in flow to rear.



5. Formulary.—*a. Sodium bicarbonate solution, 2 percent.*—This can be roughly approximated by dissolving 1 teaspoonful of sodium bicarbonate in 1 glass of water.

b. Alkali solution for prophylaxis against lewisite.

	Gm. or c. c.
Sodium hydroxide	10.0
Glycerine	30.0
Water, distilled, q. s. ad	100.0

c. Compound tannic acid powder.

	Gm. or c. c.
Potassium chloride	0.42
Calcium chloride	0.84
Salicylic acid	1.0
Sodium chloride	10.5
Tannic acid	100.0

(1) Use finely powdered materials if available, grind thoroughly and mix intimately in a mortar. Place in bottle and shake thoroughly to complete mixing.

(2) To make compound tannic acid solution, dissolve all of this powder in 1,000 c. c. of distilled water; for smaller amounts dissolve 1 teaspoonful (3 gm.) in 1 ounce (30 c. c.) of distilled water. On standing, the solution will be rendered antiseptic and stable by the salicylic acid and can be kept in the dispensary in this form.

d. Ferric hydrate paste (*Vedder*).—Add small portions of strong ammonia water successively to an aqueous solution of ferric chloride slightly under the saturation point until a slight odor of ammonia persists. Do not stir, but place in deep, narrow jars, and allow the heavy brown precipitate to settle to the bottom. Syphon off the supernatant fluid, refill with distilled water, allow precipitate to resettle, and repeat the washing process until the fluid is free from chlorides as determined by the absence of a white precipitate (AgCl) on the addition of nitric acid and silver nitrate. The washing process may require 6 to 8 days. When washing is complete, drain the material on a filter and make a thick paste by mixing 6 parts of it with 1 part of glycerine. Place in airtight ointment jars or boxes or collapsible tubes.

e. Antipruritic ointment No. 71.

	Gm. or c. c.
Butyn sulfate	0. 5
Benzyl alcohol	12. 0
Menthol	0. 2
Oil of lavender	0. 2
Alcohol	5. 0
Stearic acid	12. 0

Dissolve the butyn sulfate and menthol in the alcohol and add the benzyl alcohol and oil of lavender. Place the stearic acid in a porcelain or glass casserole, and liquefy at a temperature just below the melting point by the gentle application of heat. Add the liquid mixture slowly to the melted stearic acid, stirring continuously with a rod or pestle until gradual cooling results in solidification of the mixture. Keep in airtight ointment jars or collapsible tubes. Apply generously with rubbing to itching or painful areas of the skin.

f. Protective ointment CWS, issue.—(For protective and prophylactic action on skin against vesicants.)

g. Alcoholic sodium sulfite solution.

	Gm. or c. c.
Sodium sulfite	4. 0
Water, distilled	50. 0
Alcohol q. s. ad	100. 0

Dissolve the sodium sulfite in the water and add the alcohol. To be used as a skin wash against chlorpicrin and chloracetophenone (high concentrations or splashes).

h. Cod liver oil ointment.

	Gm. or c. c.
Cod liver oil	50. 0
Petrolatum	50. 0

Sterilize the petrolatum in an autoclave, cool, and mix thoroughly with the cod liver oil. If desired, small amounts of turpentine or tincture of myrrh may be incorporated to mask the odor of cod liver oil.

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[A. G. 062.11 (2–11–41).]

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G. C. MARSHALL,
Chief of Staff.

OFFICIAL:

E. S. ADAMS,
Major General,
The Adjutant General.

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(For explanation of symbols, see FM 21–6.)

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